

CRC(NZ) Copper Anti-Seize Aerosol

CRC Industries (CRC Industries New Zealand)

Chemwatch Hazard Alert Code: 4

Chemwatch: 4853-41

Version No: 5.1.1.1

Safety Data Sheet according to WHS and ADG requirements

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SECTION 1 IDENTIFICATION OF THE SUBSTANCE / MIXTURE AND OF THE COMPANY / UNDERTAKING

Product Identifier

Product name	CRC(NZ) Copper Anti-Seize Aerosol
Synonyms	Not Available
Proper shipping name	AEROSOLS
Other means of identification	Not Available

Relevant identified uses of the substance or mixture and uses advised against

Relevant identified uses	Lubricant grease. Application is by spray atomisation from a hand held aerosol pack Use according to manufacturer's directions.
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Details of the supplier of the safety data sheet

Registered company name	CRC Industries (CRC Industries New Zealand)
Address	10 Highbrook Drive East Tamaki Auckland New Zealand
Telephone	+64 9 272 2700
Fax	+64 9 274 9696
Website	www.crc.co.nz
Email	customerservices@crc.co.nz

Emergency telephone number

Association / Organisation	CRC Industries (CRC Industries New Zealand)
Emergency telephone numbers	NZ Poisons Centre 0800 POISON (0800 764 766)
Other emergency telephone numbers	111 (NZ Emergency Services)

SECTION 2 HAZARDS IDENTIFICATION

Classification of the substance or mixture

Poisons Schedule	Not Applicable
Classification [1]	Flammable Aerosols Category 1, Eye Irritation Category 2A, Specific target organ toxicity - single exposure Category 3 (narcotic effects), Specific target organ toxicity - repeated exposure Category 2, Aspiration Hazard Category 1, Chronic Aquatic Hazard Category 3
Legend:	1. Classified by Chemwatch; 2. Classification drawn from HCIS; 3. Classification drawn from Regulation (EU) No 1272/2008 - Annex VI

Label elements

Hazard pictogram(s)	
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SIGNAL WORD **DANGER**

Hazard statement(s)

H222	Extremely flammable aerosol.
H319	Causes serious eye irritation.
H336	May cause drowsiness or dizziness.
H373	May cause damage to organs through prolonged or repeated exposure.
H304	May be fatal if swallowed and enters airways.
H412	Harmful to aquatic life with long lasting effects.
AUH044	Risk of explosion if heated under confinement.

Precautionary statement(s) Prevention

P210	Keep away from heat/sparks/open flames/hot surfaces. - No smoking.
P211	Do not spray on an open flame or other ignition source.

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P251	Pressurized container: Do not pierce or burn, even after use.
P260	Do not breathe mist/vapours/spray.
P271	Use only outdoors or in a well-ventilated area.
P273	Avoid release to the environment.
P280	Wear protective gloves/protective clothing/eye protection/face protection.

Precautionary statement(s) Response

P301+P310	IF SWALLOWED: Immediately call a POISON CENTER or doctor/physician.
P331	Do NOT induce vomiting.
P305+P351+P338	IF IN EYES: Rinse cautiously with water for several minutes. Remove contact lenses, if present and easy to do. Continue rinsing.
P312	Call a POISON CENTER or doctor/physician if you feel unwell.
P337+P313	If eye irritation persists: Get medical advice/attention.
P304+P340	IF INHALED: Remove victim to fresh air and keep at rest in a position comfortable for breathing.

Precautionary statement(s) Storage

P405	Store locked up.
P410+P412	Protect from sunlight. Do not expose to temperatures exceeding 50 °C/122 °F.
P403+P233	Store in a well-ventilated place. Keep container tightly closed.

Precautionary statement(s) Disposal

P501	Dispose of contents/container to authorised hazardous or special waste collection point in accordance with any local regulation.
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SECTION 3 COMPOSITION / INFORMATION ON INGREDIENTS

Substances

See section below for composition of Mixtures

Mixtures

CAS No	%[weight]	Name
64742-65-0.	30-60	<u>paraffinic distillate, heavy solvent-dewaxed (severe)</u>
7782-42-5	5-10	<u>graphite</u>
7440-50-8	5-10	<u>copper</u>
68512-91-4	20-50	<u>hydrocarbons, C3-4 rich, petroleum distillate</u>

SECTION 4 FIRST AID MEASURES

Description of first aid measures

Eye Contact	<p>If aerosols come in contact with the eyes:</p> <ul style="list-style-type: none"> ▶ Immediately hold the eyelids apart and flush the eye continuously for at least 15 minutes with fresh running water. ▶ Ensure complete irrigation of the eye by keeping eyelids apart and away from eye and moving the eyelids by occasionally lifting the upper and lower lids. ▶ Transport to hospital or doctor without delay. ▶ Removal of contact lenses after an eye injury should only be undertaken by skilled personnel.
Skin Contact	<p>If solids or aerosol mists are deposited upon the skin:</p> <ul style="list-style-type: none"> ▶ Flush skin and hair with running water (and soap if available). ▶ Remove any adhering solids with industrial skin cleansing cream. ▶ DO NOT use solvents. ▶ Seek medical attention in the event of irritation.
Inhalation	<p>If aerosols, fumes or combustion products are inhaled:</p> <ul style="list-style-type: none"> ▶ Remove to fresh air. ▶ Lay patient down. Keep warm and rested. ▶ Prostheses such as false teeth, which may block airway, should be removed, where possible, prior to initiating first aid procedures. ▶ If breathing is shallow or has stopped, ensure clear airway and apply resuscitation, preferably with a demand valve resuscitator, bag-valve mask device, or pocket mask as trained. Perform CPR if necessary. ▶ Transport to hospital, or doctor.
Ingestion	<ul style="list-style-type: none"> ▶ Avoid giving milk or oils. ▶ Avoid giving alcohol. ▶ Not considered a normal route of entry.

Indication of any immediate medical attention and special treatment needed

For acute or short term repeated exposures to petroleum distillates or related hydrocarbons:

- ▶ Primary threat to life, from pure petroleum distillate ingestion and/or inhalation, is respiratory failure.
 - ▶ Patients should be quickly evaluated for signs of respiratory distress (e.g. cyanosis, tachypnoea, intercostal retraction, obtundation) and given oxygen. Patients with inadequate tidal volumes or poor arterial blood gases (pO2 50 mm Hg) should be intubated.
 - ▶ Arrhythmias complicate some hydrocarbon ingestion and/or inhalation and electrocardiographic evidence of myocardial injury has been reported; intravenous lines and cardiac monitors should be established in obviously symptomatic patients. The lungs excrete inhaled solvents, so that hyperventilation improves clearance.
 - ▶ A chest x-ray should be taken immediately after stabilisation of breathing and circulation to document aspiration and detect the presence of pneumothorax.
 - ▶ Epinephrine (adrenalin) is not recommended for treatment of bronchospasm because of potential myocardial sensitisation to catecholamines. Inhaled cardioselective bronchodilators (e.g. Alupent, Salbutamol) are the preferred agents, with aminophylline a second choice.
 - ▶ Lavage is indicated in patients who require decontamination; ensure use of cuffed endotracheal tube in adult patients. [Ellenhorn and Barceloux: Medical Toxicology]
- for copper intoxication:

- ▶ Unless extensive vomiting has occurred empty the stomach by lavage with water, milk, sodium bicarbonate solution or a 0.1% solution of potassium ferrocyanide (the resulting copper ferrocyanide is insoluble).
 - ▶ Administer egg white and other demulcents.
 - ▶ Maintain electrolyte and fluid balances.
 - ▶ Morphine or meperidine (Demerol) may be necessary for control of pain.
 - ▶ If symptoms persist or intensify (especially circulatory collapse or cerebral disturbances, try BAL intramuscularly or penicillamine in accordance with the supplier's recommendations.
 - ▶ Treat shock vigorously with blood transfusions and perhaps vasopressor amines.
 - ▶ If intravascular haemolysis becomes evident protect the kidneys by maintaining a diuresis with mannitol and perhaps by alkalinising the urine with sodium bicarbonate.
 - ▶ It is unlikely that methylene blue would be effective against the occasional methaemoglobinemia and it might exacerbate the subsequent haemolytic episode.
 - ▶ Institute measures for impending renal and hepatic failure.
- [GOSSELIN, SMITH & HODGE: Commercial Toxicology of Commercial Products]
- ▶ A role for activated charcoals or emesis is, as yet, unproven.
 - ▶ In severe poisoning CaNa₂EDTA has been proposed.

[ELLENHORN & BARCELOUX: Medical Toxicology]

Treat symptomatically.

- ▶ Heavy and persistent skin contamination over many years may lead to dysplastic changes. Pre-existing skin disorders may be aggravated by exposure to this product.
- ▶ In general, emesis induction is unnecessary with high viscosity, low volatility products, i.e. most oils and greases.
- ▶ High pressure accidental injection through the skin should be assessed for possible incision, irrigation and/or debridement.

NOTE: Injuries may not seem serious at first, but within a few hours tissue may become swollen, discoloured and extremely painful with extensive subcutaneous necrosis. Product may be forced through considerable distances along tissue planes.

SECTION 5 FIREFIGHTING MEASURES

Extinguishing media

SMALL FIRE:

- ▶ Water spray, dry chemical or CO₂

LARGE FIRE:

- ▶ Water spray or fog.

Special hazards arising from the substrate or mixture

Fire Incompatibility	▶ Avoid contamination with oxidising agents i.e. nitrates, oxidising acids, chlorine bleaches, pool chlorine etc. as ignition may result
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Advice for firefighters

Fire Fighting	<ul style="list-style-type: none"> ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water course. ▶ If safe, switch off electrical equipment until vapour fire hazard removed. ▶ Use water delivered as a fine spray to control fire and cool adjacent area. ▶ DO NOT approach containers suspected to be hot. ▶ Cool fire exposed containers with water spray from a protected location. ▶ If safe to do so, remove containers from path of fire. ▶ Equipment should be thoroughly decontaminated after use.
Fire/Explosion Hazard	<p>Combustion products include:</p> <p>carbon dioxide (CO₂)</p> <ul style="list-style-type: none"> ▶ Liquid and vapour are highly flammable. ▶ Severe fire hazard when exposed to heat or flame. ▶ Vapour forms an explosive mixture with air. ▶ Severe explosion hazard, in the form of vapour, when exposed to flame or spark. ▶ Vapour may travel a considerable distance to source of ignition. ▶ Heating may cause expansion or decomposition with violent container rupture. ▶ Aerosol cans may explode on exposure to naked flames. ▶ Rupturing containers may rocket and scatter burning materials. ▶ Hazards may not be restricted to pressure effects. ▶ May emit acrid, poisonous or corrosive fumes. ▶ On combustion, may emit toxic fumes of carbon monoxide (CO). <p>carbon monoxide (CO) phosphorus oxides (PO_x) sulfur oxides (SO_x) metal oxides other pyrolysis products typical of burning organic material.</p> <p>Contains low boiling substance: Closed containers may rupture due to pressure buildup under fire conditions.</p>
HAZCHEM	Not Applicable

SECTION 6 ACCIDENTAL RELEASE MEASURES

Personal precautions, protective equipment and emergency procedures

See section 8

Environmental precautions

See section 12

Methods and material for containment and cleaning up

Minor Spills	<p>Slippery when spilt.</p> <ul style="list-style-type: none"> ▶ Clean up all spills immediately. ▶ Avoid breathing vapours and contact with skin and eyes. ▶ Wear protective clothing, impervious gloves and safety glasses. ▶ Shut off all possible sources of ignition and increase ventilation. ▶ Wipe up.
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	<ul style="list-style-type: none"> ▶ If safe, damaged cans should be placed in a container outdoors, away from all ignition sources, until pressure has dissipated. ▶ Undamaged cans should be gathered and stowed safely.
Major Spills	<p>Slippery when spilt.</p> <ul style="list-style-type: none"> ▶ Clear area of personnel and move upwind. ▶ Alert Fire Brigade and tell them location and nature of hazard. ▶ May be violently or explosively reactive. ▶ Wear breathing apparatus plus protective gloves. ▶ Prevent, by any means available, spillage from entering drains or water courses ▶ No smoking, naked lights or ignition sources. ▶ Increase ventilation. ▶ Stop leak if safe to do so. ▶ Water spray or fog may be used to disperse / absorb vapour. ▶ Absorb or cover spill with sand, earth, inert materials or vermiculite. ▶ If safe, damaged cans should be placed in a container outdoors, away from ignition sources, until pressure has dissipated. ▶ Undamaged cans should be gathered and stowed safely. ▶ Collect residues and seal in labelled drums for disposal.

Personal Protective Equipment advice is contained in Section 8 of the SDS.

SECTION 7 HANDLING AND STORAGE

Precautions for safe handling

Safe handling	<ul style="list-style-type: none"> ▶ DO NOT allow clothing wet with material to stay in contact with skin ▶ Avoid all personal contact, including inhalation. ▶ Wear protective clothing when risk of exposure occurs. ▶ Use in a well-ventilated area. ▶ Prevent concentration in hollows and sumps. ▶ DO NOT enter confined spaces until atmosphere has been checked. ▶ Avoid smoking, naked lights or ignition sources. ▶ Avoid contact with incompatible materials. ▶ When handling, DO NOT eat, drink or smoke. ▶ DO NOT incinerate or puncture aerosol cans. ▶ DO NOT spray directly on humans, exposed food or food utensils. ▶ Avoid physical damage to containers. ▶ Always wash hands with soap and water after handling. ▶ Work clothes should be laundered separately. ▶ Use good occupational work practice. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS. ▶ Atmosphere should be regularly checked against established exposure standards to ensure safe working conditions are maintained.
Other information	<ul style="list-style-type: none"> ▶ Keep dry to avoid corrosion of cans. Corrosion may result in container perforation and internal pressure may eject contents of can ▶ Store in original containers in approved flammable liquid storage area. ▶ DO NOT store in pits, depressions, basements or areas where vapours may be trapped. ▶ No smoking, naked lights, heat or ignition sources. ▶ Keep containers securely sealed. Contents under pressure. ▶ Store away from incompatible materials. ▶ Store in a cool, dry, well ventilated area. ▶ Avoid storage at temperatures higher than 40 deg C. ▶ Store in an upright position. ▶ Protect containers against physical damage. ▶ Check regularly for spills and leaks. ▶ Observe manufacturer's storage and handling recommendations contained within this SDS.

Conditions for safe storage, including any incompatibilities

Suitable container	<ul style="list-style-type: none"> ▶ Aerosol dispenser. ▶ Check that containers are clearly labelled.
Storage incompatibility	<ul style="list-style-type: none"> ▶ Reacts with acids producing flammable / explosive hydrogen (H2) gas ▶ Avoid reaction with oxidising agents

SECTION 8 EXPOSURE CONTROLS / PERSONAL PROTECTION

Control parameters

OCCUPATIONAL EXPOSURE LIMITS (OEL)

INGREDIENT DATA

Source	Ingredient	Material name	TWA	STEL	Peak	Notes
Australia Exposure Standards	paraffinic distillate, heavy, solvent-dewaxed (severe)	Oil mist, refined mineral	5 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	graphite	Graphite (all forms except fibres) (respirable dust) (natural & synthetic)	3 mg/m3	Not Available	Not Available	(e) Containing no asbestos and < 1% crystalline silica.
Australia Exposure Standards	copper	Copper (fume)	0.2 mg/m3	Not Available	Not Available	Not Available
Australia Exposure Standards	copper	Copper, dusts & mists (as Cu)	1 mg/m3	Not Available	Not Available	Not Available

EMERGENCY LIMITS

Ingredient	Material name	TEEL-1	TEEL-2	TEEL-3
paraffinic distillate, heavy, solvent-dewaxed (severe)	Pump oil; (petroleum distillates, solvent de-waxed heavy paraffinic	140 mg/m3	1,500 mg/m3	8,900 mg/m3

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graphite	Carbon; (Graphite, 7782-42-5)	6 mg/m3	330 mg/m3	2,000 mg/m3
copper	Copper	3 mg/m3	33 mg/m3	200 mg/m3

Ingredient	Original IDLH	Revised IDLH
paraffinic distillate, heavy, solvent-dewaxed (severe)	2,500 mg/m3	Not Available
graphite	1,250 mg/m3	Not Available
copper	100 mg/m3	Not Available
hydrocarbons, C3-4 rich, petroleum distillate	Not Available	Not Available

OCCUPATIONAL EXPOSURE BANDING

Ingredient	Occupational Exposure Band Rating	Occupational Exposure Band Limit
hydrocarbons, C3-4 rich, petroleum distillate	C	> 1 to ≤ 10 parts per million (ppm)

Notes:

Occupational exposure banding is a process of assigning chemicals into specific categories or bands based on a chemical's potency and the adverse health outcomes associated with exposure. The output of this process is an occupational exposure band (OEB), which corresponds to a range of exposure concentrations that are expected to protect worker health.

MATERIAL DATA

For 1,3-butadiene:

Odour Threshold Value: 0.45 ppm (detection), 1.1 ppm (recognition)

Exposure at or below the TLV-TWA is thought to provide significant protection for workers against systemic toxicity including cancer.

US rubber workers reached an accord in 1996 to limit exposure to 1 ppm with a 15-minute, short-term limit of 5 ppm. This TLV-TWA is currently under review in light of a report of animal carcinogenicity at 6.25 ppm.

Odour Safety Factor(OSF)

OSF=1.3 ("1,3-BUTADIENE")

For liquefied petroleum gases (LPG):

TLV TWA: 1000 ppm, 1800 mg/m3 (as LPG)

ES TWA: 1000 ppm, 1800 mg/m3 (as LPG)

OES TWA: 1000 ppm, 1750 mg/m3; STEL: 1250 ppm, 2180 mg/m3 (as LPG)

IDLH Level: 2000 ppm (lower explosive limit)

No chronic systemic effects have been reported from occupational exposure to LPG. The TLV-TWA is based on good hygiene practices and is thought to minimise the risk of fire or explosion.

Odour Safety Factor(OSF)

OSF=0.16 (hydrocarbon propellant)

For graphite:

Graphite pneumoconiosis resembles coal workers' pneumoconiosis. Data indicate that the higher the crystalline silica content of graphite the more likely the disease will increase in severity. The presence of anthracite coal in the production of some synthetic grades of graphite appears to make arbitrary the use of the term, "synthetic", "artificial" or "natural".

The TLV-TWA for carbon black is recommended to minimise complaints of excessive dirtiness and applies only to commercially produced carbon blacks or to soots derived from combustion sources containing absorbed polycyclic aromatic hydrocarbons (PAHs). When PAHs are present in carbon black (measured as the cyclohexane-extractable fraction) NIOSH has established a REL-TWA of 0.1 mg/m3 and considers the material to be an occupational carcinogen.

The NIOSH REL-TWA was "selected on the basis of professional judgement rather than on data delineating safe from unsafe concentrations of PAHs".

This limit was justified on the basis of feasibility of measurement and not on a demonstration of its safety.

NOTE H: Special requirements exist in relation to classification and labelling of this substance. This note applies to certain coal- and oil -derived substances and to certain entries for groups of substances in Annex VI. European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

NOTE L: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 3% DMSO extract as measured by IP 346.


European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

NOTE K: The classification as a carcinogen need not apply if it can be shown that the substance contains less than 0.1%w/w 1,3-butadiene (EINECS No 203-450-8). - European Union (EU) List of harmonised classification and labelling hazardous substances, Table 3.1, Annex VI, Regulation (EC) No 1272/2008 (CLP) - up to the latest ATP

Exposure controls

Appropriate engineering controls	<p>Engineering controls are used to remove a hazard or place a barrier between the worker and the hazard. Well-designed engineering controls can be highly effective in protecting workers and will typically be independent of worker interactions to provide this high level of protection.</p> <p>The basic types of engineering controls are:</p> <p>Process controls which involve changing the way a job activity or process is done to reduce the risk.</p> <p>Enclosure and/or isolation of emission source which keeps a selected hazard "physically" away from the worker and ventilation that strategically "adds" and "removes" air in the work environment. Ventilation can remove or dilute an air contaminant if designed properly. The design of a ventilation system must match the particular process and chemical or contaminant in use.</p> <p>Employers may need to use multiple types of controls to prevent employee overexposure.</p> <ul style="list-style-type: none"> ▶ Employees exposed to confirmed human carcinogens should be authorized to do so by the employer, and work in a regulated area. ▶ Work should be undertaken in an isolated system such as a "glove-box". Employees should wash their hands and arms upon completion of the assigned task and before engaging in other activities not associated with the isolated system. ▶ Within regulated areas, the carcinogen should be stored in sealed containers, or enclosed in a closed system, including piping systems, with any sample ports or openings closed while the carcinogens are contained within. ▶ Open-vessel systems are prohibited. ▶ Each operation should be provided with continuous local exhaust ventilation so that air movement is always from ordinary work areas to the operation. ▶ Exhaust air should not be discharged to regulated areas, non-regulated areas or the external environment unless decontaminated. Clean make-up air should be introduced in sufficient volume to maintain correct operation of the local exhaust system. ▶ For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. ▶ Except for outdoor systems, regulated areas should be maintained under negative pressure (with respect to non-regulated areas). ▶ Local exhaust ventilation requires make-up air be supplied in equal volumes to replaced air. ▶ Laboratory hoods must be designed and maintained so as to draw air inward at an average linear face velocity of 0.76 m/sec with a minimum
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	<p>of 0.64 m/sec. Design and construction of the fume hood requires that insertion of any portion of the employees body, other than hands and arms, be disallowed.</p> <p>Exhaust ventilation should be designed to prevent accumulation and recirculation in the workplace and safely remove carbon black from the air.</p> <p>Note: Wet, activated carbon removes oxygen from the air and thus presents a severe hazard to workers inside carbon vessels and enclosed or confined spaces. Before entering such areas sampling and test procedures for low oxygen levels should be undertaken and control conditions set up to ensure ample oxygen availability.[Linde]</p>
Personal protection	
Eye and face protection	<p>No special equipment for minor exposure i.e. when handling small quantities.</p> <p>OTHERWISE: For potentially moderate or heavy exposures:</p> <ul style="list-style-type: none"> ▶ Safety glasses with side shields. ▶ NOTE: Contact lenses pose a special hazard; soft lenses may absorb irritants and ALL lenses concentrate them.
Skin protection	See Hand protection below
Hands/feet protection	<p>NOTE:</p> <ul style="list-style-type: none"> ▶ The material may produce skin sensitisation in predisposed individuals. Care must be taken, when removing gloves and other protective equipment, to avoid all possible skin contact. ▶ Contaminated leather items, such as shoes, belts and watch-bands should be removed and destroyed. ▶ No special equipment needed when handling small quantities. <p>OTHERWISE:</p> <ul style="list-style-type: none"> ▶ For potentially moderate exposures: ▶ Wear general protective gloves, eg. light weight rubber gloves. ▶ For potentially heavy exposures: ▶ Wear chemical protective gloves, eg. PVC. and safety footwear.
Body protection	See Other protection below
Other protection	<ul style="list-style-type: none"> ▶ Employees working with confirmed human carcinogens should be provided with, and be required to wear, clean, full body protective clothing (smocks, coveralls, or long-sleeved shirt and pants), shoe covers and gloves prior to entering the regulated area. [AS/NZS ISO 6529:2006 or national equivalent] ▶ Employees engaged in handling operations involving carcinogens should be provided with, and required to wear and use half-face filter-type respirators with filters for dusts, mists and fumes, or air purifying canisters or cartridges. A respirator affording higher levels of protection may be substituted. [AS/NZS 1715 or national equivalent] ▶ Emergency deluge showers and eyewash fountains, supplied with potable water, should be located near, within sight of, and on the same level with locations where direct exposure is likely. ▶ Prior to each exit from an area containing confirmed human carcinogens, employees should be required to remove and leave protective clothing and equipment at the point of exit and at the last exit of the day, to place used clothing and equipment in impervious containers at the point of exit for purposes of decontamination or disposal. The contents of such impervious containers must be identified with suitable labels. For maintenance and decontamination activities, authorized employees entering the area should be provided with and required to wear clean, impervious garments, including gloves, boots and continuous-air supplied hood. ▶ Prior to removing protective garments the employee should undergo decontamination and be required to shower upon removal of the garments and hood. <p>No special equipment needed when handling small quantities.</p> <p>OTHERWISE:</p> <ul style="list-style-type: none"> ▶ Overalls. ▶ Skin cleansing cream. ▶ Eyewash unit. ▶ Do not spray on hot surfaces. ▶ The clothing worn by process operators insulated from earth may develop static charges far higher (up to 100 times) than the minimum ignition energies for various flammable gas-air mixtures. This holds true for a wide range of clothing materials including cotton. ▶ Avoid dangerous levels of charge by ensuring a low resistivity of the surface material worn outermost. <p>BRETHERRICK: Handbook of Reactive Chemical Hazards.</p>

Respiratory protection

Type A-P Filter of sufficient capacity. (AS/NZS 1716 & 1715, EN 143:2000 & 149:2001, ANSI Z88 or national equivalent)

Where the concentration of gas/particulates in the breathing zone, approaches or exceeds the "Exposure Standard" (or ES), respiratory protection is required. Degree of protection varies with both face-piece and Class of filter; the nature of protection varies with Type of filter.

Required Minimum Protection Factor	Half-Face Respirator	Full-Face Respirator	Powered Air Respirator
up to 10 x ES	A-AUS P2	-	A-PAPR-AUS / Class 1 P2
up to 50 x ES	-	A-AUS / Class 1 P2	-
up to 100 x ES	-	A-2 P2	A-PAPR-2 P2 ^

^ - Full-face

A(All classes) = Organic vapours, B AUS or B1 = Acid gasses, B2 = Acid gas or hydrogen cyanide(HCN), B3 = Acid gas or hydrogen cyanide(HCN), E = Sulfur dioxide(SO2), G = Agricultural chemicals, K = Ammonia(NH3), Hg = Mercury, NO = Oxides of nitrogen, MB = Methyl bromide, AX = Low boiling point organic compounds(below 65 degC)

- ▶ Cartridge respirators should never be used for emergency ingress or in areas of unknown vapour concentrations or oxygen content.
- ▶ The wearer must be warned to leave the contaminated area immediately on detecting any odours through the respirator. The odour may indicate that the mask is not functioning properly, that the vapour concentration is too high, or that the mask is not properly fitted. Because of these limitations, only restricted use of cartridge respirators is considered appropriate.
- ▶ Cartridge performance is affected by humidity. Cartridges should be changed after 2 hr of continuous use unless it is determined that the humidity is less than 75%, in which case, cartridges can be used for 4 hr. Used cartridges should be discarded daily, regardless of the length of time used

SECTION 9 PHYSICAL AND CHEMICAL PROPERTIES

Information on basic physical and chemical properties

Appearance	Copper coloured paste with a characteristic odour; not miscible with water. Supplied as an aerosol pack. Contents under PRESSURE . Contains highly flammable hydrocarbon propellant.
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Physical state	Liquid	Relative density (Water = 1)	Not Available
Odour	Not Available	Partition coefficient n-octanol / water	Not Available
Odour threshold	Not Available	Auto-ignition temperature (°C)	Not Available
pH (as supplied)	Not Available	Decomposition temperature	Not Available
Melting point / freezing point (°C)	Not Available	Viscosity (cSt)	Not Available
Initial boiling point and boiling range (°C)	Not Available	Molecular weight (g/mol)	Not Applicable
Flash point (°C)	-60 (propellant)	Taste	Not Available
Evaporation rate	Not Available	Explosive properties	Not Available
Flammability	HIGHLY FLAMMABLE.	Oxidising properties	Not Available
Upper Explosive Limit (%)	Not Available	Surface Tension (dyn/cm or mN/m)	Not Available
Lower Explosive Limit (%)	Not Available	Volatile Component (%vol)	Not Available
Vapour pressure (kPa)	Not Available	Gas group	Not Available
Solubility in water	Immiscible	pH as a solution (1%)	Not Available
Vapour density (Air = 1)	Not Available	VOC g/L	Not Available

SECTION 10 STABILITY AND REACTIVITY

Reactivity	See section 7
Chemical stability	<ul style="list-style-type: none"> ▶ Elevated temperatures. ▶ Presence of open flame. ▶ Product is considered stable. ▶ Hazardous polymerisation will not occur. ▶ Presence of heat source ▶ Presence of an ignition source
Possibility of hazardous reactions	See section 7
Conditions to avoid	See section 7
Incompatible materials	See section 7
Hazardous decomposition products	See section 5

SECTION 11 TOXICOLOGICAL INFORMATION

Information on toxicological effects

	<p>Inhalation of vapours may cause drowsiness and dizziness. This may be accompanied by narcosis, reduced alertness, loss of reflexes, lack of coordination and vertigo.</p> <p>Inhalation of aerosols (mists, fumes), generated by the material during the course of normal handling, may be damaging to the health of the individual.</p> <p>Limited evidence or practical experience suggests that the material may produce irritation of the respiratory system, in a significant number of individuals, following inhalation. In contrast to most organs, the lung is able to respond to a chemical insult by first removing or neutralising the irritant and then repairing the damage. The repair process, which initially evolved to protect mammalian lungs from foreign matter and antigens, may however, produce further lung damage resulting in the impairment of gas exchange, the primary function of the lungs. Respiratory tract irritation often results in an inflammatory response involving the recruitment and activation of many cell types, mainly derived from the vascular system.</p> <p>Common, generalised symptoms associated with toxic gas inhalation include:</p> <ul style="list-style-type: none"> ▶ central nervous system effects such as depression, headache, confusion, dizziness, progressive stupor, coma and seizures; ▶ respiratory system complications may include acute pulmonary oedema, dyspnoea, stridor, tachypnoea, bronchospasm, wheezing and other reactive airway symptoms, and respiratory arrest; ▶ cardiovascular effects may include cardiovascular collapse, arrhythmias and cardiac arrest; ▶ gastrointestinal effects may also be present and may include mucous membrane irritation, nausea and vomiting (sometimes bloody), and abdominal pain.
Inhaled	<p>Inhalation hazard is increased at higher temperatures.</p> <p>Central nervous system (CNS) depression may include nonspecific discomfort, symptoms of giddiness, headache, dizziness, nausea, anaesthetic effects, slowed reaction time, slurred speech and may progress to unconsciousness. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>Inhalation of oil droplets/ aerosols may cause discomfort and may produce chemical pneumonitis.</p> <p>Material is highly volatile and may quickly form a concentrated atmosphere in confined or unventilated areas. The vapour may displace and replace air in breathing zone, acting as a simple asphyxiant. This may happen with little warning of overexposure.</p> <p>Acute effects from inhalation of high concentrations of vapour are pulmonary irritation, including coughing, with nausea; central nervous system depression - characterised by headache and dizziness, increased reaction time, fatigue and loss of co-ordination</p> <p>WARNING: Intentional misuse by concentrating/inhaling contents may be lethal.</p> <p>High inhaled concentrations of mixed hydrocarbons may produce narcosis characterised by nausea, vomiting and lightheadedness. Inhalation of aerosols may produce severe pulmonary oedema, pneumonitis and pulmonary haemorrhage. Inhalation of petroleum hydrocarbons consisting substantially of low molecular weight species (typically C2-C12) may produce irritation of mucous membranes, incoordination, giddiness, nausea, vertigo, confusion, headache, appetite loss, drowsiness, tremors and anaesthetic stupor. Massive exposures may produce central nervous system depression with sudden collapse and deep coma; fatalities have been recorded. Irritation of the brain and/or apnoeic anoxia may produce convulsions. Although recovery following overexposure is generally complete, cerebral micro-haemorrhage of focal post-inflammatory scarring may produce epileptiform seizures some months after the exposure. Pulmonary episodes may include chemical pneumonitis with oedema and haemorrhage. The lighter hydrocarbons may produce kidney and neurotoxic effects. Pulmonary irritancy increases with carbon chain length for</p>

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	<p>paraffins and olefins. Alkenes produce pulmonary oedema at high concentrations. Liquid paraffins may produce anaesthesia and depressant actions leading to weakness, dizziness, slow and shallow respiration, unconsciousness, convulsions and death. C5-7 paraffins may also produce polyneuropathy. Aromatic hydrocarbons accumulate in lipid rich tissues (typically the brain, spinal cord and peripheral nerves) and may produce functional impairment manifested by nonspecific symptoms such as nausea, weakness, fatigue and vertigo; severe exposures may produce inebriation or unconsciousness. Many of the petroleum hydrocarbons are cardiac sensitizers and may cause ventricular fibrillations. Hydrocarbons may sensitise the heart to adrenalin and other circulatory catecholamines; as a result cardiac arrhythmias and ventricular fibrillation may occur. Abrupt collapse may produce traumatic injury. Central nervous system (CNS) depression may be evident early. Symptoms of moderate poisoning may include giddiness, headache, dizziness and nausea. Serious poisonings may result in respiratory depression and may be fatal.</p> <p>The paraffin gases C1-4 are practically non-toxic below their lower flammability limits (18000-50000 ppm). Above this level, incidental effects include CNS depression and irritation but these are reversible upon cessation of the exposure. The C3 and iso-C5 hydrocarbons show increasing narcotic properties; branching of the chain also enhances the effect. The C4 hydrocarbons appear to be more highly neurotoxic than the C3 and C5 members. Several fatalities due to voluntary inhalation of butane have been reported, possibly due to central, respiratory and circulatory effects resulting from anaesthesia, laryngeal oedema, chemical pneumonia or the combined effects of cardiac toxicity and increased sympathomimetic effects.</p> <p>Inhalation of petroleum gases may produce narcosis, due in part to olefinic impurities. Displacement of oxygen in the air may cyanosis. If present in sufficient quantity these gases may reduce the oxygen level to below 18% producing asphyxiation. Symptoms include rapid respiration, mental dullness, lack of coordination, poor judgement, nausea and vomiting. The onset of cyanosis may lead to unconsciousness and death.</p>
Ingestion	<p>Accidental ingestion of the material may be damaging to the health of the individual. Not normally a hazard due to physical form of product. Considered an unlikely route of entry in commercial/industrial environments Ingestion of petroleum hydrocarbons may produce irritation of the pharynx, oesophagus, stomach and small intestine with oedema and mucosal ulceration resulting; symptoms include a burning sensation in the mouth and throat. Large amounts may produce narcosis with nausea and vomiting, weakness or dizziness, slow and shallow respiration, swelling of the abdomen, unconsciousness and convulsions. Myocardial injury may produce arrhythmias, ventricular fibrillation and electrocardiographic changes. Central nervous system depression may also occur. Light aromatic hydrocarbons produce a warm, sharp, tingling sensation on contact with taste buds and may anaesthetise the tongue. Aspiration into the lungs may produce coughing, gagging and a chemical pneumonitis with pulmonary oedema and haemorrhage.</p> <p>Numerous cases of a single oral exposure to high levels of copper have been reported. Consumption of copper-contaminated drinking water has been associated with mainly gastrointestinal symptoms including nausea, abdominal pain, vomiting and diarrhoea. A metallic taste, nausea, vomiting and epigastric burning often occur after ingestion of copper and its derivatives. The vomitus is usually green/blue and discolours contaminated skin. Acute poisonings from the ingestion of copper salts are rare due to their prompt removal by vomiting. Vomiting is due mainly to the local and astringent action of copper ion on the stomach and bowel. Emesis usually occurs within 5 to 10 minutes but may be delayed if food is present in the stomach. Should vomiting not occur, or is delayed, gradual absorption from the bowel may result in systemic poisoning with death, possibly, following within several days. Apparent recovery may be followed by lethal relapse. Systemic effects of copper resemble other heavy metal poisonings and produce wide-spread capillary damage, kidney and liver damage and central nervous system excitation followed by depression. Haemolytic anaemia (a result of red-blood cell damage) has been described in acute human poisoning. [GOSSELLIN, SMITH HODGE: Clinical Toxicology of Commercial Products.]</p> <p>Other symptoms of copper poisoning include lethargy, neurotoxicity, and increased blood pressure and respiratory rates. Coma and death have followed attempted suicides using solutions of copper sulfate. Copper is an essential element and most animal tissues have measurable amounts of copper associated with them. Humans have evolved mechanisms which maintain its availability whilst limiting its toxicity (homeostasis). Copper is initially bound in the body to a blood-borne protein, serum albumin and thereafter is more firmly bound to another protein, alpha-ceruloplasmin. Such binding effectively "inactivates" the copper, thus reducing its potential to produce toxic damage. In healthy individuals, bound copper can reach relatively high levels without producing adverse health effects. Excretion in the bile represents the major pathway by which copper is removed from the body when it reaches potentially toxic levels. Copper may also be stored in the liver and bone marrow where it is bound to another protein, metallothionein. A combination of binding and excretion ensures that the body is able to tolerate relatively high loadings of copper.</p>
Skin Contact	<p>Repeated exposure may cause skin cracking, flaking or drying following normal handling and use.</p> <p>Limited evidence exists, or practical experience predicts, that the material either produces inflammation of the skin in a substantial number of individuals following direct contact, and/or produces significant inflammation when applied to the healthy intact skin of animals, for up to four hours, such inflammation being present twenty-four hours or more after the end of the exposure period. Skin irritation may also be present after prolonged or repeated exposure; this may result in a form of contact dermatitis (nonallergic). The dermatitis is often characterised by skin redness (erythema) and swelling (oedema) which may progress to blistering (vesiculation), scaling and thickening of the epidermis. At the microscopic level there may be intercellular oedema of the spongy layer of the skin (spongiosis) and intracellular oedema of the epidermis. Spray mist may produce discomfort</p> <p>Open cuts, abraded or irritated skin should not be exposed to this material</p> <p>Exposure to copper, by skin, has come from its use in pigments, ointments, ornaments, jewellery, dental amalgams and IUDs and as an antifungal agent and an algicide. Although copper algicides are used in the treatment of water in swimming pools and reservoirs, there are no reports of toxicity from these applications. Reports of allergic contact dermatitis following contact with copper and its salts have appeared in the literature, however the exposure concentrations leading to any effect have been poorly characterised. In one study, patch testing of 1190 eczema patients found that only 13 (1.1%) cross-reacted with 2% copper sulfate in petrolatum. The investigators warned, however, that the possibility of contamination with nickel (an established contact allergen) might have been the cause of the reaction. Copper salts often produce an itching eczema in contact with skin. This is, likely, of a non-allergic nature.</p> <p>The material may accentuate any pre-existing dermatitis condition</p>
Eye	<p>Limited evidence exists, or practical experience suggests, that the material may cause eye irritation in a substantial number of individuals and/or is expected to produce significant ocular lesions which are present twenty-four hours or more after instillation into the eye(s) of experimental animals. Repeated or prolonged eye contact may cause inflammation characterised by temporary redness (similar to windburn) of the conjunctiva (conjunctivitis); temporary impairment of vision and/or other transient eye damage/ulceration may occur.</p> <p>Direct contact with the eye may not cause irritation because of the extreme volatility of the gas; however concentrated atmospheres may produce irritation after brief exposures..</p> <p>Petroleum hydrocarbons may produce pain after direct contact with the eyes. Slight, but transient disturbances of the corneal epithelium may also result. The aromatic fraction may produce irritation and lachrymation.</p> <p>Copper salts, in contact with the eye, may produce conjunctivitis or even ulceration and turbidity of the cornea.</p>
Chronic	<p>Practical experience shows that skin contact with the material is capable either of inducing a sensitisation reaction in a substantial number of individuals, and/or of producing a positive response in experimental animals.</p> <p>On the basis of epidemiological data, the material is regarded as carcinogenic to humans. There is sufficient data to establish a causal association between human exposure to the material and the development of cancer.</p> <p>There is sufficient evidence to provide a strong presumption that human exposure to the material may result in the development of heritable genetic damage, generally on the basis of</p> <ul style="list-style-type: none"> - appropriate animal studies, - other relevant information <p>Harmful: danger of serious damage to health by prolonged exposure through inhalation.</p> <p>Serious damage (clear functional disturbance or morphological change which may have toxicological significance) is likely to be caused by</p>

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repeated or prolonged exposure. As a rule the material produces, or contains a substance which produces severe lesions. Such damage may become apparent following direct application in subchronic (90 day) toxicity studies or following sub-acute (28 day) or chronic (two-year) toxicity tests.

Limited evidence suggests that repeated or long-term occupational exposure may produce cumulative health effects involving organs or biochemical systems.

There is some evidence that human exposure to the material may result in developmental toxicity. This evidence is based on animal studies where effects have been observed in the absence of marked maternal toxicity, or at around the same dose levels as other toxic effects but which are not secondary non-specific consequences of the other toxic effects.

Principal route of occupational exposure to the gas is by inhalation.

Chronic copper poisoning is rarely recognised in man although in one instance, at least, symptoms more commonly associated with exposures to mercury, namely infantile acro-dynia (pink disease), have been described. Tissue damage of mucous membranes may follow chronic dust exposure. A hazardous situation is exposure of a worker with the rare hereditary condition (Wilson's disease or hereditary hepatolenticular degeneration) to copper exposure which may cause liver, kidney, CNS, bone and sight damage and is potentially lethal. Haemolytic anaemia (a result of red-blood cell damage) is common in cows and sheep poisoned by copper derivatives. Overdosing of copper feed supplements has resulted in pigmentary cirrhosis of the liver. [GOSSELIN, SMITH HODGE: Clinical Toxicology of Commercial Products]

Repeated or prolonged exposure to mixed hydrocarbons may produce narcosis with dizziness, weakness, irritability, concentration and/or memory loss, tremor in the fingers and tongue, vertigo, olfactory disorders, constriction of visual field, paraesthesias of the extremities, weight loss and anaemia and degenerative changes in the liver and kidney. Chronic exposure by petroleum workers, to the lighter hydrocarbons, has been associated with visual disturbances, damage to the central nervous system, peripheral neuropathies (including numbness and paraesthesias), psychological and neurophysiological deficits, bone marrow toxicities (including hypoplasia possibly due to benzene) and hepatic and renal involvement. Chronic dermal exposure to petroleum hydrocarbons may result in defatting which produces localised dermatoses.

Surface cracking and erosion may also increase susceptibility to infection by microorganisms. One epidemiological study of petroleum refinery workers has reported elevations in standard mortality ratios for skin cancer along with a dose-response relationship indicating an association between routine workplace exposure to petroleum or one of its constituents and skin cancer, particularly melanoma. Other studies have been unable to confirm this finding.

Hydrocarbon solvents are liquid hydrocarbon fractions derived from petroleum processing streams, containing only carbon and hydrogen atoms, with carbon numbers ranging from approximately C5-C20 and boiling between approximately 35-370 deg C. Many of the hydrocarbon solvents have complex and variable compositions with constituents of 4 types, alkanes (normal paraffins, isoparaffins, and cycloparaffins) and aromatics (primarily alkylated one- and two-ring species). Despite the compositional complexity, most hydrocarbon solvent constituents have similar toxicological properties, and the overall toxicological hazards can be characterized in generic terms. Hydrocarbon solvents can cause chemical pneumonitis if aspirated into the lung, and those that are volatile can cause acute CNS effects and/or ocular and respiratory irritation at exposure levels exceeding occupational recommendations. Otherwise, there are few toxicologically important effects. The exceptions, n-hexane and naphthalene, have unique toxicological properties

Animal studies:

No deaths or treatment related signs of toxicity were observed in rats exposed to light alkylate naphtha (paraffinic hydrocarbons) at concentrations of 668, 2220 and 6646 ppm for 6 hrs/day, 5 days/wk for 13 weeks. Increased liver weights and kidney toxicity (male rats) was observed in high dose animals. Exposure to pregnant rats at concentrations of 137, 3425 and 6850 ppm did not adversely affect reproduction or cause maternal or foetal toxicity. Lifetime skin painting studies in mice with similar naphthas have shown weak or no carcinogenic activity following prolonged and repeated exposure. Similar

naphthas/distillates, when tested at nonirritating dose levels, did not show any significant carcinogenic activity indicating that this tumorigenic response is likely related to chronic irritation and not to dose. The mutagenic potential of naphthas has been reported to be largely negative in a variety of mutagenicity tests. The exact relationship between these results and human health is not known. Some components of this product have been shown to produce a species specific, sex hormonal dependent kidney lesion in male rats from repeated oral or inhalation exposure. Subsequent research has shown that the kidney damage develops via the formation of a alpha-2u-globulin, a mechanism unique to the male rat. Humans do not form alpha-2u-globulin, therefore, the kidney effects resulting from this mechanism are not relevant in human.

Principal route of exposure is by skin contact; lesser exposures include inhalation of fumes from hot oils, oil mists or droplets. Prolonged contact with mineral oils carries with it the risk of skin conditions such as oil folliculitis, eczematous dermatitis, pigmentation of the face (melanosis) and warts on the sole of the foot (plantar warts). With highly refined mineral oils no appreciable systemic effects appear to result through skin absorption.

Exposure to oil mists frequently elicits respiratory conditions, such as asthma; the provoking agent is probably an additive. High oil mist concentrations may produce lipid pneumonia although clinical evidence is equivocal. In animals exposed to concentrations of 100 mg/m3 oil mist, for periods of 12 to 26 months, the activity of lung and serum alkaline phosphatase enzyme was raised; 5 mg/m3 oil mist did not produce this response. These enzyme changes are sensitive early indicators of lung damage. Workers exposed to vapours of mineral oil and kerosene for 5 to 35 years showed an increased prevalence of slight basal lung fibrosis.

CRC(NZ) Copper Anti-Seize Aerosol	TOXICITY	IRRITATION
	Not Available	Not Available
paraffinic distillate, heavy, solvent-dewaxed (severe)	TOXICITY	IRRITATION
	Dermal (rabbit) LD50: >2000 mg/kg ^[2]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (rat) LC50: >5.3 mg/l4 h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (rat) LD50: >5000 mg/kg ^[2]	
graphite	TOXICITY	IRRITATION
	Inhalation (rat) LC50: >2 mg/l4 h ^[1]	Not Available
	Oral (rat) LD50: >2000 mg/kg ^[2]	
copper	TOXICITY	IRRITATION
	dermal (rat) LD50: >2000 mg/kg ^[1]	Eye: no adverse effect observed (not irritating) ^[1]
	Inhalation (rat) LC50: 0.733 mg/l4 h ^[1]	Skin: no adverse effect observed (not irritating) ^[1]
	Oral (rat) LD50: 300-500 mg/kg ^[1]	
hydrocarbons, C3-4 rich, petroleum distillate	TOXICITY	IRRITATION
	Not Available	Not Available

Legend:

1. Value obtained from Europe ECHA Registered Substances - Acute toxicity 2. * Value obtained from manufacturer's SDS. Unless otherwise specified data extracted from RTECS - Register of Toxic Effect of chemical Substances

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PARAFFINIC DISTILLATE, HEAVY, SOLVENT-DEWAXED (SEVERE)	<p>Studies indicate that normal, branched and cyclic paraffins are absorbed from the mammalian gastrointestinal tract and that the absorption of n-paraffins is inversely proportional to the carbon chain length, with little absorption above C30. With respect to the carbon chain lengths likely to be present in mineral oil, n-paraffins may be absorbed to a greater extent than iso- or cyclo-paraffins.</p> <p>The major classes of hydrocarbons have been shown to be well absorbed by the gastrointestinal tract in various species. In many cases, the hydrophobic hydrocarbons are ingested in association with dietary lipids. The dependence of hydrocarbon absorption on concomitant triglyceride digestion and absorption, is known as the "hydrocarbon continuum hypothesis", and asserts that a series of solubilising phases in the intestinal lumen, created by dietary triglycerides and their digestion products, afford hydrocarbons a route to the lipid phase of the intestinal absorptive cell (enterocyte) membrane. While some hydrocarbons may traverse the mucosal epithelium unmetabolised and appear as solutes in lipoprotein particles in intestinal lymph, there is evidence that most hydrocarbons partially separate from nutrient lipids and undergo metabolic transformation in the enterocyte. The enterocyte may play a major role in determining the proportion of an absorbed hydrocarbon that, by escaping initial biotransformation, becomes available for deposition in its unchanged form in peripheral tissues such as adipose tissue, or in the liver.</p> <p>The substance is classified by IARC as Group 3: NOT classifiable as to its carcinogenicity to humans. Evidence of carcinogenicity may be inadequate or limited in animal testing.</p>
GRAPHITE	<p>Asthma-like symptoms may continue for months or even years after exposure to the material ceases. This may be due to a non-allergic condition known as reactive airways dysfunction syndrome (RADS) which can occur following exposure to high levels of highly irritating compound. Key criteria for the diagnosis of RADS include the absence of preceding respiratory disease, in a non-atopic individual, with abrupt onset of persistent asthma-like symptoms within minutes to hours of a documented exposure to the irritant. A reversible airflow pattern, on spirometry, with the presence of moderate to severe bronchial hyperreactivity on methacholine challenge testing and the lack of minimal lymphocytic inflammation, without eosinophilia, have also been included in the criteria for diagnosis of RADS. RADS (or asthma) following an irritating inhalation is an infrequent disorder with rates related to the concentration of and duration of exposure to the irritating substance. Industrial bronchitis, on the other hand, is a disorder that occurs as result of exposure due to high concentrations of irritating substance (often particulate in nature) and is completely reversible after exposure ceases. The disorder is characterised by dyspnea, cough and mucus production.</p>
COPPER	<p>WARNING: Inhalation of high concentrations of copper fume may cause "metal fume fever", an acute industrial disease of short duration. Symptoms are tiredness, influenza like respiratory tract irritation with fever.</p> <p>for copper and its compounds (typically copper chloride):</p> <p>Acute toxicity: There are no reliable acute oral toxicity results available. In an acute dermal toxicity study (OECD TG 402), one group of 5 male rats and 5 groups of 5 female rats received doses of 1000, 1500 and 2000 mg/kg bw via dermal application for 24 hours. The LD50 values of copper monochloride were 2,000 mg/kg bw or greater for male (no deaths observed) and 1,224 mg/kg bw for female. Four females died at both 1500 and 2000 mg/kg bw, and one at 1,000 mg/kg bw. Symptom of the hardness of skin, an exudation of hardness site, the formation of scar and reddish changes were observed on application sites in all treated animals. Skin inflammation and injury were also noted. In addition, a reddish or black urine was observed in females at 2,000, 1,500 and 1,000 mg/kg bw. Female rats appeared to be more sensitive than male based on mortality and clinical signs.</p> <p>No reliable skin/eye irritation studies were available. The acute dermal study with copper monochloride suggests that it has a potential to cause skin irritation.</p> <p>Repeat dose toxicity: In repeated dose toxicity study performed according to OECD TG 422, copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39 - 51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL value was 5 and 1.3 mg/kg bw/day for male and female rats, respectively. No deaths were observed in male rats. One treatment-related death was observed in female rats in the high dose group. Erythropoietic toxicity (anaemia) was seen in both sexes at the 80 mg/kg bw/day. The frequency of squamous cell hyperplasia of the forestomach was increased in a dose-dependent manner in male and female rats at all treatment groups, and was statistically significant in males at doses of =20 mg/kg bw/day and in females at doses of =5 mg/kg bw/day doses. The observed effects are considered to be local, non-systemic effect on the forestomach which result from oral (gavage) administration of copper monochloride.</p> <p>Genotoxicity: An in vitro genotoxicity study with copper monochloride showed negative results in a bacterial reverse mutation test with Salmonella typhimurium strains (TA 98, TA 100, TA 1535, and TA 1537) with and without S9 mix at concentrations of up to 1,000 ug/plate. An in vitro test for chromosome aberration in Chinese hamster lung (CHL) cells showed that copper monochloride induced structural and numerical aberrations at the concentration of 50, 70 and 100 ug/mL without S9 mix. In the presence of the metabolic activation system, significant increases of structural aberrations were observed at 50 and 70 ug/mL and significant increases of numerical aberrations were observed at 70 ug/mL. In an in vivo mammalian erythrocyte micronucleus assay, all animals dosed (15 - 60 mg/kg bw) with copper monochloride exhibited similar PCE/(PCE+NCE) ratios and MNPCE frequencies compared to those of the negative control animals. Therefore copper monochloride is not an in vivo mutagen.</p> <p>Carcinogenicity: there was insufficient information to evaluate the carcinogenic activity of copper monochloride.</p> <p>Reproductive and developmental toxicity: In the combined repeated dose toxicity study with the reproduction/developmental toxicity screening test (OECD TG 422), copper monochloride was given orally (gavage) to Sprague-Dawley rats for 30 days to males and for 39-51 days to females at concentrations of 0, 1.3, 5.0, 20, and 80 mg/kg bw/day. The NOAEL of copper monochloride for fertility toxicity was 80 mg/kg bw/day for the parental animals. No treatment-related effects were observed on the reproductive organs and the fertility parameters assessed. For developmental toxicity the NOAEL was 20 mg/kg bw/day. Three of 120 pups appeared to have icterus at birth; 4 of 120 pups appeared runted at the highest dose tested (80 mg/kg bw/day).</p>
CRC(NZ) Copper Anti-Seize Aerosol & PARAFFINIC DISTILLATE, HEAVY, SOLVENT-DEWAXED (SEVERE) & GRAPHITE & HYDROCARBONS, C3-4 RICH, PETROLEUM DISTILLATE	<p>No significant acute toxicological data identified in literature search.</p>
CRC(NZ) Copper Anti-Seize Aerosol & HYDROCARBONS, C3-4 RICH, PETROLEUM DISTILLATE	<p>for Petroleum Hydrocarbon Gases:</p> <p>In many cases, there is more than one potentially toxic constituent in a refinery gas. In those cases, the constituent that is most toxic for a particular endpoint in an individual refinery stream is used to characterize the endpoint hazard for that stream. The hazard potential for each mammalian endpoint for each of the petroleum hydrocarbon gases is dependent upon each petroleum hydrocarbon gas constituent endpoint toxicity values (LC50, LOAEL, etc.) and the relative concentration of the constituent present in that gas. It should also be noted that for an individual petroleum hydrocarbon gas, the constituent characterizing toxicity may be different for different mammalian endpoints, again, being dependent upon the concentration of the different constituents in each, distinct petroleum hydrocarbon gas.</p> <p>All Hydrocarbon Gases Category members contain primarily hydrocarbons (i.e., alkanes and alkenes) and occasionally asphyxiant gases like hydrogen. The inorganic components of the petroleum hydrocarbon gases are less toxic than the C1 - C4 and C5 - C6 hydrocarbon components to both mammalian and aquatic organisms. Unlike other petroleum product categories (e.g. gasoline, diesel fuel, lubricating oils, etc.), the inorganic and hydrocarbon constituents of hydrocarbon gases can be evaluated for hazard individually to then predict the screening level hazard</p>

of the Category members

Acute toxicity: No acute toxicity LC50 values have been derived for the C1 -C4 and C5- C6 hydrocarbon (HC) fractions because no mortality was observed at the highest exposure levels tested (~ 5 mg/l) for these petroleum hydrocarbon gas constituents. The order of acute toxicity of petroleum hydrocarbon gas constituents from most to least toxic is:

C5-C6 HCs (LC50 > 1063 ppm) > C1-C4 HCs (LC50 > 10,000 ppm) > benzene (LC50 = 13,700 ppm) > butadiene (LC50 = 129,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).

Repeat dose toxicity: With the exception of the asphyxiant gases, repeated dose toxicity has been observed in individual selected petroleum hydrocarbon gas constituents. Based upon LOAEL values, the order of order of repeated-dose toxicity of these constituents from most toxic to the least toxic is:

Benzene (LOAEL .>=10 ppm) >C1-C4 HCs (LOAEL = 5,000 ppm; assumed to be 100% 2-butene) > C5-C6 HCs (LOAEL = 6,625 ppm) > butadiene (LOAEL = 8,000 ppm) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).

Genotoxicity:

In vitro: The majority of the Petroleum Hydrocarbon Gases Category components are negative for *in vitro* genotoxicity. The exceptions are: benzene and 1,3-butadiene, which are genotoxic in bacterial and mammalian *in vitro* test systems.

In vivo: The majority of the Petroleum Hydrocarbon Gases Category components are negative for *in vivo* genotoxicity. The exceptions are benzene and 1,3-butadiene, which are genotoxic in *in vivo* test systems

Developmental toxicity: Developmental effects were induced by two of the petroleum hydrocarbon gas constituents, benzene and the C5 -C6 hydrocarbon fraction. No developmental toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for developmental toxicity. Based on LOAEL and NOAEL values, the order of acute toxicity of these constituents from most to least toxic is:

Benzene (LOAEL = 20 ppm) > butadiene (NOAEL .>=1,000 ppm) > C5-C6 HCs (LOAEL = 3,463 ppm) > C1-C4 HCs (NOAEL >=5,000 ppm; assumed to be 100% 2-butene) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen).

Reproductive toxicity: Reproductive effects were induced by only two petroleum hydrocarbon gas constituents, benzene and isobutane (a constituent of the the C1-C4 hydrocarbon fraction). No reproductive toxicity was observed at the highest exposure levels tested for the other petroleum hydrocarbon gas constituents tested for this effect. The asphyxiant gases have not been tested for reproductive toxicity. Based on LOAEL and NOAEL values, the order of reproductive toxicity of these constituents from most to least toxic is:

Benzene (LOAEL = 300 ppm) > butadiene (NOAEL .>=6,000 ppm) > C5-C6 HCs (NOAEL .>=6,521 ppm) > C1-C4 HCs (LOAEL = 9,000 ppm; assumed to be 100% isobutane) > asphyxiant gases (hydrogen, carbon dioxide, nitrogen)

The materials included in the Lubricating Base Oils category are related from both process and physical-chemical perspectives;

The potential toxicity of a specific distillate base oil is inversely related to the severity or extent of processing the oil has undergone, since:

- The adverse effects of these materials are associated with undesirable components, and
- The levels of the undesirable components are inversely related to the degree of processing;
- Distillate base oils receiving the same degree or extent of processing will have similar toxicities;
- The potential toxicity of *residual base oils* is independent of the degree of processing the oil receives.
- The reproductive and developmental toxicity of the distillate base oils is inversely related to the degree of processing.

The degree of refining influences the carcinogenic potential of the oils. Whereas mild acid / earth refining processes are inadequate to substantially reduce the carcinogenic potential of lubricant base oils, hydrotreatment and / or solvent extraction methods can yield oils with no carcinogenic potential.

Unrefined and mildly refined distillate base oils contain the highest levels of undesirable components, have the largest variation of hydrocarbon molecules and have shown the highest potential carcinogenic and mutagenic activities. Highly and severely refined distillate base oils are produced from unrefined and mildly refined oils by removing or transforming undesirable components. In comparison to unrefined and mildly refined base oils, the highly and severely refined distillate base oils have a smaller range of hydrocarbon molecules and have demonstrated very low mammalian toxicity. Mutagenicity and carcinogenicity testing of residual oils has been negative, supporting the belief that these materials lack biologically active components or the components are largely non-bioavailable due to their molecular size.

Toxicity testing has consistently shown that lubricating base oils have low acute toxicities. Numerous tests have shown that a lubricating base oil's mutagenic and carcinogenic potential correlates with its 3-7 ring polycyclic aromatic compound (PAC) content, and the level of DMSO extractables (e.g. IP346 assay), both characteristics that are directly related to the degree/conditions of processing

Skin irritating is not significant (CONCAWE) based on 14 tests on 10 CASs from the OLBO class (Other Lubricant Base Oils). Each study lasted for 24 hours, a period of time 6 times longer than the duration recommended by the OECD method).

Eye irritation is not significant according to experimental data (CONCAWE studies) based on 9 "in vivo" tests on 7 CASs from the OLBO class(Other Lubricant Base Oils).

Sensitisation: The substance does not cause the sensitization of the respiratory tract or of the skin. (CONCAWE studies based on 14 tests on 11 CASs from the OLBO class(Other Lubricant Base Oils))

Germ cell mutagenicity: The tests performed within the "in vivo" studies regarding gene mutation at mice micronuclei indicated negative results (CONCAWE studies. AMES tests had negative results in 7 studies performed on 4 CASs from the OLBO class(Other Lubricant Base Oils)).

Reproduction toxicity: Reproduction / development toxicity monitoring according to OECD 421 or 422 methods. CONCAWE tests gave negative results in oral gavage studies. Pre-birth studies regarding toxicity in the unborn foetus development process showed a maternal LOAEL (Lowest Observed Adverse Effect Level) of 125 mg/kg body/day, based on dermal irritation and a NOAEL (No Observable Adverse Effect Level) of 2000 mg/kg body/day, which shows that the substance is not toxic for reproduction.

STOT (toxicity on specific target organs) – repeated exposure: Studies with short term repeated doses (28-day test) on rabbit skin indicated the NOAEL value of 1000 mg/kg. NOAEL for inhalation, local effects > 280 mg/m3 and for systemic effects NOAEL > 980 mg/m3.

Sub-chronic toxicity

90-day study Dermal: NOAEL > 2000 mg/kg (CONCAWE studies).

Repeat dose toxicity:

Oral

NOAEL for heavy paraffinic distillate aromatic extract could not be identified and is less than 125 mg/kg/day when administered orally.

Inhalation

The NOAEL for lung changes associated with oil deposition in the lungs was 220 mg/m3. As no systemic toxicity was observed, the overall NOAEL for systemic effects was > 980 mg/m3.

Dermal

In a 90 day subchronic dermal study, the administration of Light paraffinic distillate solvent extract had an adverse effect on survivability, body weights, organ weights (particularly the liver and thymus), and variety of haematology and serum chemistry parameters in exposed animals.

Histopathological changes which were treatment-related were most prominent in the adrenals, bone marrow, kidneys, liver, lymph nodes, skin, stomach, and thymus. Based on the results of this study, the NOAEL for the test material is less than 30 mg/kg/day.

Toxicity to reproduction:

Mineral oil (a white mineral oil) caused no reproductive or developmental toxicity with 1 mL/kg/day (i.e., 1000 mg/kg/day) in an OECD 421 guideline study, but did cause mild to moderate skin irritation. Therefore, the reproductive/developmental NOAEL for this study is =1000 mg/kg/day and no LOAEL was determined.

Developmental toxicity, teratogenicity:

Heavy paraffinic distillate furfural extract produced maternal, reproductive and foetal toxicity. Maternal toxicity was exhibited as vaginal discharge (dose-related), body weight decrease, reduction in thymus weight and increase in liver weight (125 mg/kg/day and higher) and aberrant haematology and serum chemistry (125 and/or 500 mg/kg/day). Evidence of potential reproductive effects was shown by an increased number of dams with resorptions and intrauterine death. Distillate aromatic extract (DAE) was developmentally toxic regardless of exposure duration as indicated by increased resorptions and decreased foetal body weights. Furthermore, when exposures were increased to 1000 mg/kg/day and given only during gestation days 10 through 12, cleft palate and ossification delays were observed. Cleft palate was considered to indicate a potential teratogenic effect of DAE.

The following Oil Industry Note (OIN) has been applied: OIN 8 - The classifications as a reproductive toxicant category 2; H361d (Suspected of

CRC(NZ) Copper Anti-Seize
Aerosol & PARAFFINIC
DISTILLATE, HEAVY,
SOLVENT-DEWAXED
(SEVERE)

CRC(NZ) Copper Anti-Seize Aerosol

damaging the unborn child) and specific target organ toxicant category 1; H372 (Causes damage to organs through prolonged or repeated exposure) need not apply if the substance is not classified as carcinogenic

Toxicokinetics of lubricant base oils has been examined in rodents. Absorption of other lubricant base oils across the small intestine is related to carbon chain length; hydrocarbons with smaller chain length are more readily absorbed than hydrocarbons with a longer chain length. The majority of an oral dose of mineral hydrocarbon is not absorbed and is excreted unchanged in the faeces. Distribution of mineral hydrocarbons following absorption has been observed in liver, fat, kidney, brain and spleen. Excretion of absorbed mineral hydrocarbons occurs via the faeces and urine. Based on the pharmacokinetic parameters and disposition profiles, the data indicate inherent strain differences in the total systemic exposure (~4 fold greater systemic dose in F344 vs SD rats), rate of metabolism, and hepatic and lymph node retention of C26H52, which may be associated with the different strain sensitivities to the formation of liver granulomas and MLN histiocytosis.

Highly and Severely Refined Distillate Base Oils

Acute toxicity: Multiple studies of the acute toxicity of highly & severely refined base oils have been reported. Irrespective of the crude source or the method or extent of processing, the oral LD50s have been observed to be >5 g/kg (bw) and the dermal LD50s have ranged from >2 to >5g/kg (bw). The LC50 for inhalation toxicity ranged from 2.18 mg/l to > 4 mg/l.

When tested for skin and eye irritation, the materials have been reported as "non-irritating" to "moderately irritating"

Testing in guinea pigs for sensitization has been negative

Repeat dose toxicity: Several studies have been conducted with these oils. The weight of evidence from all available data on highly & severely refined base oils support the presumption that a distillate base oil's toxicity is inversely related to the degree of processing it receives. Adverse effects have been reported with even the most severely refined white oils - these appear to depend on animal species and/ or the peculiarities of the study.

- ▶ The granulomatous lesions induced by the oral administration of white oils are essentially foreign body responses. The lesions occur only in rats, of which the Fischer 344 strain is particularly sensitive,
- ▶ The testicular effects seen in rabbits after dermal administration of a highly to severely refined base oil were unique to a single study and may have been related to stress induced by skin irritation, and
- ▶ The accumulation of foamy macrophages in the alveolar spaces of rats exposed repeatedly via inhalation to high levels of highly to severely refined base oils is not unique to these oils, but would be seen after exposure to many water insoluble materials.

Reproductive and developmental toxicity: A highly refined base oil was used as the vehicle control in a one-generation reproduction study.

The study was conducted according to the OECD Test Guideline 421. There was no effect on fertility and mating indices in either males or females. At necropsy, there were no consistent findings and organ weights and histopathology were considered normal by the study's authors. A single generation study in which a white mineral oil (a food/ drug grade severely refined base oil) was used as a vehicle control is reported.

Two separate groups of pregnant rats were administered 5 ml/kg (bw)/day of the base oil via gavage, on days 6 through 19 of gestation. In one of the two base oil dose groups, three malformed foetuses were found among three litters. The study authors considered these malformations to be minor and within the normal ranges for the strain of rat.

Genotoxicity:

In vitro (mutagenicity): Several studies have reported the results of testing different base oils for mutagenicity using a modified Ames assay. Base oils with no or low concentrations of 3-7 ring PACs had low mutagenicity indices.

In vivo (chromosomal aberrations): A total of seven base stocks were tested in male and female Sprague-Dawley rats using a bone marrow cytogenetics assay. The test materials were administered via gavage at dose levels ranging from 500 to 5000 mg/kg (bw). Dosing occurred for either a single day or for five consecutive days. None of the base oils produced a significant increase in aberrant cells.

Carcinogenicity: Highly & severely refined base oils are not carcinogens, when given either orally or dermally.

Acute Toxicity	✗	Carcinogenicity	✗
Skin Irritation/Corrosion	✗	Reproductivity	✗
Serious Eye Damage/Irritation	✓	STOT - Single Exposure	✓
Respiratory or Skin sensitisation	✗	STOT - Repeated Exposure	✓
Mutagenicity	✗	Aspiration Hazard	✓

Legend: ✗ – Data either not available or does not fill the criteria for classification
 ✓ – Data available to make classification

SECTION 12 ECOLOGICAL INFORMATION

Toxicity

CRC(NZ) Copper Anti-Seize Aerosol	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	Not Available	Not Available	Not Available	Not Available	Not Available
paraffinic distillate, heavy, solvent-dewaxed (severe)	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>10-mg/L	2
	EC50	96	Algae or other aquatic plants	>1000mg/L	1
graphite	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	>100mg/L	2
	EC50	48	Crustacea	>100mg/L	2
	EC50	72	Algae or other aquatic plants	>100mg/L	2
copper	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	0.001-0.09mg/L	2
	EC50	48	Crustacea	0.001mg/L	2
	EC50	72	Algae or other aquatic plants	0.013335mg/L	4
	BCF	960	Fish	200mg/L	4

Continued...

CRC(NZ) Copper Anti-Seize Aerosol

	EC25	6	Algae or other aquatic plants	0.00150495mg/L	4
	NOEC	96	Crustacea	0.0008mg/L	4
hydrocarbons, C3-4 rich, petroleum distillate	ENDPOINT	TEST DURATION (HR)	SPECIES	VALUE	SOURCE
	LC50	96	Fish	24.11mg/L	2
	EC50	96	Algae or other aquatic plants	7.71mg/L	2

Legend: *Extracted from 1. IUCLID Toxicity Data 2. Europe ECHA Registered Substances - Ecotoxicological Information - Aquatic Toxicity 3. EPIWIN Suite V3.12 (QSAR) - Aquatic Toxicity Data (Estimated) 4. US EPA, Ecotox database - Aquatic Toxicity Data 5. ECETOC Aquatic Hazard Assessment Data 6. NITE (Japan) - Bioconcentration Data 7. METI (Japan) - Bioconcentration Data 8. Vendor Data*

Harmful to aquatic organisms, may cause long-term adverse effects in the aquatic environment.

Do NOT allow product to come in contact with surface waters or to intertidal areas below the mean high water mark. Do not contaminate water when cleaning equipment or disposing of equipment wash-waters.

Wastes resulting from use of the product must be disposed of on site or at approved waste sites.

When spilled this product may act as a typical oil, causing a film, sheen, emulsion or sludge at or beneath the surface of the body of water. The oil film on water surface may physically affect the aquatic organisms, due to the interruption of the oxygen transfer between the air and the water

Oils of any kind can cause:

- ▶ drowning of water-fowl due to lack of buoyancy, loss of insulating capacity of feathers, starvation and vulnerability to predators due to lack of mobility
- ▶ lethal effects on fish by coating gill surfaces, preventing respiration
- ▶ asphyxiation of benthic life forms when floating masses become engaged with surface debris and settle on the bottom and
- ▶ adverse aesthetic effects of fouled shoreline and beaches

In case of accidental releases on the soil, a fine film is formed on the soil, which prevents the plant respiration process and the soil particle saturation. It may cause deep water infestation.

Metal-containing inorganic substances generally have negligible vapour pressure and are not expected to partition to air. Once released to surface waters and moist soils their fate depends on solubility and dissociation in water. Environmental processes (such as oxidation and the presence of acids or bases) may transform insoluble metals to more soluble ionic forms. Microbiological processes may also transform insoluble metals to more soluble forms. Such ionic species may bind to dissolved ligands or sorb to solid particles in aquatic or aqueous media. A significant proportion of dissolved/ sorbed metals will end up in sediments through the settling of suspended particles. The remaining metal ions can then be taken up by aquatic organisms.

When released to dry soil most metals will exhibit limited mobility and remain in the upper layer; some will leach locally into ground water and/ or surface water ecosystems when soaked by rain or melt ice. Environmental processes may also be important in changing solubilities.

Even though many metals show few toxic effects at physiological pHs, transformation may introduce new or magnified effects.

A metal ion is considered infinitely persistent because it cannot degrade further.

The current state of science does not allow for an unambiguous interpretation of various measures of bioaccumulation.

The counter-ion may also create health and environmental concerns once isolated from the metal. Under normal physiological conditions the counter-ion may be essentially insoluble and may not be bioavailable.

Environmental processes may enhance bioavailability.

for Petroleum Hydrocarbon Gases:

Environmental fate:

The environmental fate characteristics of petroleum hydrocarbon gases are governed by these physical-chemical attributes. All components of these gases will partition to the air where interaction with hydroxyl radicals is an important fate process. Hydrocarbons having molecular weights represented in these streams are inherently biodegradable, but their tendency to partition to the atmosphere would prevent their biotic degradation in water and soils. However, if higher molecular weight fractions of these streams enter the aquatic or terrestrial environment, biodegradation may be an important fate mechanism.

The majority of components making up hydrocarbon gases typically have low melting and boiling points. They also have high vapor pressures and low octanol/water partition coefficients. The aqueous solubilities of these substances vary, and range from approximately 22 parts per million to several hundred parts per million. The environmental fate characteristics of refinery gases are governed by these physical-chemical attributes. Components of the hydrocarbon gas streams will partition to the air, and photodegradation reactions will be an important fate process for many of the hydrocarbon components. The hydrocarbons in these mixtures are inherently biodegradable, but due to their tendency to partition to the atmosphere, biodegradation is not anticipated to be an important fate mechanisms. However, if released to water or soil, some of the higher molecular weight fractions may become available for microbial attack. The inorganic gases are chemically stable and may be lost to the atmosphere or simply become involved in the environmental recycling of their atoms. Some show substantial water solubility, but their volatility eventually causes these gases to enter the atmosphere.

Substances in Refinery Gases that volatilise to air may undergo a gas-phase oxidation reaction with photochemically produced hydroxyl radicals (OH⁻). Atmospheric oxidation as a result of hydroxyl radical attack is not direct photochemical degradation, but rather indirect degradation. Indirect photodegradation of the hydrocarbon components in Refinery Gases can be an important fate process for these constituents. In general, half lives decrease with increasing carbon chain length. Half lives for this fraction of Refinery Gases ranged from 960 days (methane) to 0.16 days (butadiene). The constituents of the C5- C6 hydrocarbon fraction have photodegradation half-lives of approximately two days.

The hydrocarbon and non-hydrocarbon constituents in Refinery Gases do not contain the functional groups or chemical linkages known to undergo hydrolysis reactions. Therefore hydrolysis will not play an important role in the environmental fate for the components in Refinery Gas streams.

Biodegradation of the hydrocarbon components in refinery gases may occur in soil and water. Gaseous hydrocarbons are widespread in nature and numerous types of microbes have evolved which are capable of oxidizing these substances as their sole energy source. Although volatilization is the predominant behavior for these gases, sufficient aqueous solubility and bioavailability is exhibited by these compounds. The use of gaseous carbon sources for cell growth is common among autotrophic organisms. Higher chain length hydrocarbons typical of naphtha streams also are known to inherently biodegrade in the environment

Ecotoxicity:

Acute LC/EC50 values for the hydrocarbon components of these gas streams ranged roughly from 1 to 100 mg/L.

Although the LC/EC50 data for the individual gases illustrate the potential toxicity to aquatic organisms, aqueous concentrations from releases of these gases would likely not persist in the aquatic environment for a sufficient duration to elicit toxicity. Based on a simple conceptual exposure model analysis, emissions of petroleum hydrocarbon gases to the atmosphere would not likely result in acutely toxic concentrations in adjacent water bodies because such emissions will tend to remain in the atmosphere.

Several of the constituents in refinery gases were shown to be highly hazardous to aquatic organisms in laboratory toxicity tests where exposure concentrations can be maintained over time. Hydrogen sulfide was shown to be the most toxic constituent to fish (LC50 ranged 0.007 to 0.2 mg/L) and invertebrates (EC50 ranged 0.022 to 1.07 mg/L), although several LC/EC50 values for ammonia also were below 1 mg/l for these organisms (0.083 to 4.6 mg/L and 0.53 to 22.8 mg/L, respectively).

for lubricating oil base stocks:

Vapor Pressure Vapor pressures of lubricating base oils are reported to be negligible. In one study, the experimentally measured vapour pressure of a solvent-dewaxed heavy paraffinic distillate base oil was 1.7 x 10exp-4 Pa. Since base oils are mixtures of C15 to C50 paraffinic, naphthenic, and aromatic hydrocarbon isomers, representative components of those structures were selected to calculate a range of vapor pressures. The estimated vapor pressure values for these selected components of base oils ranged from 4.5 x 10exp-1 Pa to 2 x 10exp-13Pa. Based on Dalton's Law the expected total vapour pressure for base oils would fall well below minimum levels (10exp-5 Pa) of recommended experimental procedures.

Partition Coefficient (log Kow): In mixtures such as the base oils, the percent distribution of the hydrocarbon groups (i.e., paraffins, naphthenes, and aromatics) and the carbon chain lengths determines in-part the partitioning characteristics of the mixture. Generally, hydrocarbon chains with fewer carbon atoms tend to have lower partition coefficients than those with higher carbon numbers. However, due to their complex composition, unequivocal determination of the log Kow of these hydrocarbon mixtures cannot be made. Rather, partition coefficients of selected C15 chain-length hydrocarbon structures representing paraffinic, naphthenic, and aromatic constituents in base oil lubricants were modelled. Results showed typical log Kow values from 4.9 to 7.7, which were consistent with values of >4 for lubricating oil basestocks

Water Solubility:When released to water, base oils will float and spread at a rate that is viscosity dependent. While water solubility of base oils is typically very low, individual hydrocarbons exhibit a wide range of solubility depending on molecular weight and degree of unsaturation. Decreasing molecular weight (i.e., carbon number) and increasing levels of unsaturation increases the water solubility of these materials. As noted for partition coefficient, the water solubility of lubricating base oils cannot be determined due to their complex mixture characteristics. Therefore, the water solubility of individual C15 hydrocarbons representing the different groups making up base oils (i.e., linear and branched paraffins, naphthenes, and aromatics) was modelled. Based on water solubility modelling of those groups, aqueous solubilities are typically much less than 1 ppm. (0.003-0.63 mg/l)

Environmental Fate:

Photodegradation: Chemicals having potential to photolyse have UV/visible absorption maxima in the range of 290 to 800 nm. Some chemicals have absorption maxima significantly below 290 nm and consequently cannot undergo direct photolysis in sunlight (e.g. chemicals such as alkanes, alkenes, alkynes, saturated alcohols, and saturated acids). Most hydrocarbon constituents of the materials in this category are not expected to photolyse since they do not show absorbance within the 290-800 nm range. However, photodegradation of polyaromatic hydrocarbons (PAHs) can occur and may be a significant degradation pathway for these constituents of lubricating base oils. The degree and rate at which PAHs may photodegrade depend upon whether conditions allow penetration of light with sufficient energy to effect a change. For example, polycyclic aromatic compounds (PAC) compounds bound to sediments may persist due to a lack of sufficient light penetration.

Atmospheric gas-phase reactions can occur between organic chemicals and reactive molecules such as photochemically produced hydroxyl radicals, ozone and nitrogen oxides. Atmospheric oxidation as a result of radical attack is not direct photochemical degradation, but indirect degradation. In general, lubricating base oils have low vapour pressures and volatilisation is not expected to be a significant removal mechanism for the majority of the hydrocarbon components. However, some components (e.g., C15 branched paraffins and naphthenes) appear to have the potential to volatilise. Atmospheric half-lives of 0.10 to 0.66 days have been calculated for representative C15 hydrocarbon components of lubricating base oils.

Stability in Water: Chemicals that have a potential to hydrolyze include alkyl halides, amides, carbamates, carboxylic acid esters and lactones, epoxides, phosphate esters, and sulfonic acid esters. Because lubricating base oils do not contain significant levels of these functional groups, materials in the lubricating base oils category are not subject to hydrolysis.

Chemical Transport and Distribution in the Environment : Based on the physical-chemical characteristics of component hydrocarbons in lubricating base oils, the lower molecular weight components are expected to have the highest vapour pressures and water solubilities, and the lowest partition coefficients. These factors enhance the potential for widespread distribution in the environment. To gain an understanding of the potential transport and distribution of lubricating base oil components, the EQC (Equilibrium Criterion) model was used to characterize the environmental distribution of different C15 compounds representing different structures found in lube oils (e.g., paraffins, naphthenes, and aromatics). The modelling found partitioning to soil or air is the ultimate fate of these C15 compounds. Aromatic compounds partition principally to soil. Linear paraffins partition mostly to soil, while branching appears to allow greater distribution to air. Naphthenes distribute to both soil and air, with increasing proportions in soil for components with the greater number of ring structures. Because the modelling does not take into account degradation factors, levels modelled in the atmosphere are likely overstated in light of the tendency for indirect photodegradation to occur.

Biodegradation: The extent of biodegradation measured for a particular lubricating oil basestock is dependent not only on the procedure used but also on how the sample is presented in the biodegradation test. Lubricant base oils typically are not readily biodegradable in standard 28-day tests. However, since the oils consist primarily of hydrocarbons that are ultimately assimilated by microorganisms, and therefore inherently biodegradable. Twenty-eight biodegradability studies have been reported for a variety of lubricating base oils. Based on the results of ultimate biodegradability tests using modified Sturm and manometric respirometry testing the base oils are expected to be, for the most part, inherently biodegradable. Biodegradation rates found using the modified Sturm procedure ranged from 1.5 to 29%. Results from the manometric respirometry tests on similar materials showed biodegradation rates from 31 to 50%. Biodegradation rates measured in 21-day CEC tests for similar materials ranged from 13 to 79%.

Ecotoxicity:

Numerous acute studies covering fish, invertebrates, and algae have been conducted to assess the ecotoxicity of various lubricating base oils. None of these studies have shown evidence of acute toxicity to aquatic organisms. Eight, 7-day exposure studies using rainbow trout failed to demonstrate toxicity when tested up to the maximum concentration of 1000 mg/L applied as dispersions. Three, 96-hour tests with rainbow trout also failed to show any toxic effects when tested up to 1000 mg/L applied as dispersions. Similarly, three 96-hour tests with fathead minnows at a maximum test concentration of 100 mg/L water accommodated fractions (WAF) showed no adverse effects. Two species of aquatic invertebrates (*Daphnia magna* and *Gammarus* sp.) were exposed to WAF solutions up to 10,000 mg/L for 48 and 96-hours, respectively, with no adverse effects being observed. Four-day exposures of the freshwater green alga (*Scenedesmus subspicatus*) to 500 mg/L WAF solutions failed to show adverse effects on growth rate and algal cell densities in four studies. Multiple chronic ecotoxicity studies have shown no adverse effects to daphnid survival or reproduction. In 10 of 11 chronic studies, daphnids were exposed for 21 days to WAF preparations of lubricating base oils with no ill effects on survival or reproduction at the maximum concentration of 1000 mg/L. One test detected a reduction in reproduction at 1000 mg/L. Additional data support findings of no chronic toxicity to aquatic invertebrates and fish. No observed effect levels ranged from 550 to 5,000 mg/L when tested as either dispersions or WAFs.

The data described above are supported by studies on a homologous series of alkanes. The author concluded that the water solubility of carbon chains .C10 is too limited to elicit acute toxicity. This also was shown for alkylbenzene compounds having carbon numbers .C15. Since base oils consist of carbon compounds of C15 to C50, component hydrocarbons that are of acute toxicological concern are, for the most part, absent in these materials. Similarly, due to their low solubility, the alkylated two to three ring polyaromatic components in base oils are not expected to cause acute or chronic toxicity. This lack of toxicity is borne out in the results of the reported studies.

The effects of crude and refined oils on organisms found in fresh and sea water has been extensively reviewed.

sea water. Where spillages occur the non-mobile species suffer the greatest mortality, whereas fish species can often escape from the affected region. The extent of the initial mortality depends on the chemical nature of the oil, the location, and the physical conditions, particularly the temperature and wind velocity. Most affected freshwater and marine communities recover from the effects of an oil spill within a year. The occurrence of biogenic hydrocarbons in the world's oceans is well recorded. They have the characteristic isoprenoid structure, and measurements made in water columns indicate a background concentration of 1.0 to 10 µl/l. The higher molecular weight materials are dispersed as particles, with the highest concentrations of about 20 µl/l occurring in the top 3 mm layer of water.

A wide variation in the response of organisms to oil exposures has been noted. The larvae of fish and crustaceans appear to be most susceptible to the water-soluble fraction of crude oil. Exposures of plankton and algae have indicated that certain species of diatoms and green algae are inhibited, whereas microflagellates are not.

For the most part, molluscs and most intertidal worm species appear to be tolerant of oil contamination.

Copper is unlikely to accumulate in the atmosphere due to a short residence time for airborne copper aerosols. Airborne coppers, however, may be transported over large distances.

Copper accumulates significantly in the food chain.

Drinking Water Standards:

3000 µg/l (UK max)

2000 µg/l (WHO provisional Guideline)

1000 µg/l (WHO level where individuals complain)

Soil Guidelines: Dutch Criteria

36 mg/kg (target)

190 mg/kg (intervention)

Air Quality Standards: no data available.

The toxic effect of copper in the aquatic biota depends on the bio-availability of copper in water which, in turn, depends on its physico-chemical form (ie.speciation). Bioavailability is decreased by complexation and adsorption of copper by natural organic matter, iron and manganese hydrated oxides, and chelating agents excreted by algae and other aquatic organisms. Toxicity is also affected by pH and hardness. Total copper is rarely useful as a predictor of toxicity. In natural sea water, more than 98% of copper is organically bound and in river waters a high percentage is often organically bound, but the actual percentage depends on the river water and its pH.

Copper exhibits significant toxicity in some aquatic organisms. Some algal species are very sensitive to copper with EC50 (96 hour) values as low as 47 µg/litre dissolved copper whilst for other algal species EC50 values of up to 481 µg/litre have been reported. However many of the reportedly high EC50 values may arise in experiments conducted with a culture media containing copper-complexing agents such as silicate, iron, manganese and EDTA which reduce bioavailability.

Toxic effects arising following exposure by aquatic species to copper are typically:

Algae EC50 (96 h)	Daphnia magna LC50 (48-96 h)	Amphipods LC50 (48-96 h)	Gastropods LC50 (48-96 h)	Crab larvae LC50 (48-96 h)
47-481 *	7-54 *	37-183 *	58-112 *	50-100 *

* µg/litre

Exposure to concentrations ranging from one to a few hundred micrograms per litre has led to sublethal effects and effects on long-term survival. For high bioavailability waters, effect concentrations for several sensitive species may be below 10 µg Cu/litre.

In fish, the acute lethal concentration of copper ranges from a few µg/litre to several mg/litre, depending both on test species and exposure conditions. Where the value is less than 50 µg Cu/litre, test waters generally have a low dissolved organic carbon (DOC) level, low hardness and neutral to slightly acidic pH. Exposure to concentrations ranging from one to a few hundred micrograms per litre has led to sublethal effects and effects on long-term survival. Lower effect concentrations are generally associated with test waters of high bioavailability.

In summary:

Responses expected for high concentration ranges of copper *
Total dissolved Cu Effects of high availability in water

concentration range (ug/litre)	
1-10	Significant effects are expected for diatoms and sensitive invertebrates, notably cladocerans. Effects on fish could be significant in freshwaters with low pH and hardness.
10-100	Significant effects are expected on various species of microalgae, some species of macroalgae, and a range of invertebrates, including crustaceans, gastropods and sea urchins. Survival of sensitive fish will be affected and a variety of fish show sublethal effects.
100-1000	Most taxonomic groups of macroalgae and invertebrates will be severely affected. Lethal levels for most fish species will be reached.
>1000	Lethal concentrations for most tolerant organisms are reached.

* Sites chosen have moderate to high bioavailability similar to water used in most toxicity tests.

Substances containing unsaturated carbons are ubiquitous in indoor environments. They result from many sources (see below). Most are reactive with environmental ozone and many produce stable products which are thought to adversely affect human health. The potential for surfaces in an enclosed space to facilitate reactions should be considered.

Source of unsaturated substances	Unsaturated substances (Reactive Emissions)	Major Stable Products produced following reaction with ozone.
Occupants (exhaled breath, ski oils, personal care products)	Isoprene, nitric oxide, squalene, unsaturated sterols, oleic acid and other unsaturated fatty acids, unsaturated oxidation products	Methacrolein, methyl vinyl ketone, nitrogen dioxide, acetone, 6MHQ, geranyl acetone, 4OPA, formaldehyde, nonanol, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid.
Soft woods, wood flooring, including cypress, cedar and silver fir boards, houseplants	Isoprene, limonene, alpha-pinene, other terpenes and sesquiterpenes	Formaldehyde, 4-AMC, pinoaldehyde, pinic acid, pinonic acid, formic acid, methacrolein, methyl vinyl ketone, SOAs including ultrafine particles
Carpets and carpet backing	4-Phenylcyclohexene, 4-vinylcyclohexene, styrene, 2-ethylhexyl acrylate, unsaturated fatty acids and esters	Formaldehyde, acetaldehyde, benzaldehyde, hexanal, nonanal, 2-nonenal
Linoleum and paints/polishes containing linseed oil	Linoleic acid, linolenic acid	Propanal, hexanal, nonanal, 2-heptenal, 2-nonenal, 2-decenal, 1-pentene-3-one, propionic acid, n-butyric acid
Latex paint	Residual monomers	Formaldehyde
Certain cleaning products, polishes, waxes, air fresheners	Limonene, alpha-pinene, terpinolene, alpha-terpineol, linalool, linalyl acetate and other terpenoids, longifolene and other sesquiterpenes	Formaldehyde, acetaldehyde, glycoaldehyde, formic acid, acetic acid, hydrogen and organic peroxides, acetone, benzaldehyde, 4-hydroxy-4-methyl-5-hexen-1-ol, 5-ethenyl-dihydro-5-methyl-2(3H)-furanone, 4-AMC, SOAs including ultrafine particles
Natural rubber adhesive	Isoprene, terpenes	Formaldehyde, methacrolein, methyl vinyl ketone
Photocopier toner, printed paper, styrene polymers	Styrene	Formaldehyde, benzaldehyde
Environmental tobacco smoke	Styrene, acrolein, nicotine	Formaldehyde, benzaldehyde, hexanal, glyoxal, N-methylformamide, nicotinaldehyde, cotinine
Soiled clothing, fabrics, bedding	Squalene, unsaturated sterols, oleic acid and other saturated fatty acids	Acetone, geranyl acetone, 6MHO, 4OPA, formaldehyde, nonanal, decanal, 9-oxo-nonanoic acid, azelaic acid, nonanoic acid
Soiled particle filters	Unsaturated fatty acids from plant waxes, leaf litter, and other vegetative debris; soot; diesel particles	Formaldehyde, nonanal, and other aldehydes; azelaic acid; nonanoic acid; 9-oxo-nonanoic acid and other oxo-acids; compounds with mixed functional groups (=O, -OH, and -COOH)
Ventilation ducts and duct liners	Unsaturated fatty acids and esters, unsaturated oils, neoprene	C5 to C10 aldehydes
"Urban grime"	Polycyclic aromatic hydrocarbons	Oxidized polycyclic aromatic hydrocarbons
Perfumes, colognes, essential oils (e.g. lavender, eucalyptus, tea tree)	Limonene, alpha-pinene, linalool, linalyl acetate, terpinene-4-ol, gamma-terpinene	Formaldehyde, 4-AMC, acetone, 4-hydroxy-4-methyl-5-hexen-1-ol, 5-ethenyl-dihydro-5-methyl-2(3H) furanone, SOAs including ultrafine particles
Overall home emissions	Limonene, alpha-pinene, styrene	Formaldehyde, 4-AMC, pinaldehyde, acetone, pinic acid, pinonic acid, formic acid, benzaldehyde, SOAs including ultrafine particles

Abbreviations: 4-AMC, 4-acetyl-1-methylcyclohexene; 6MHQ, 6-methyl-5-heptene-2-one, 4OPA, 4-oxopentanal, SOA, Secondary Organic Aerosols

Reference: Charles J Weschler; Environmental Health Perspectives, Vol 114, October 2006

In soil, copper levels are raised by application of fertiliser, fungicides, from deposition of highway dusts and from urban, mining and industrial sources. Generally, vegetation rooted in soils reflects the soil copper levels in its foliage. This is dependent upon the bioavailability of copper and the physiological requirements of species concerned.

Typical foliar levels of copper are:

Uncontaminated soils (0.3-250 mg/kg)

6.1-25 mg/kg

Contaminated soils (150-450 mg/kg)

80 mg/kg

Mining/smelter soils

300 mg/kg

Plants rarely show symptoms of toxicity or of adverse growth effects at normal soil concentrations of copper. Crops are often more sensitive to copper than the native flora, so protection levels for agricultural crops range from 25 mg Cu/kg to several hundred mg/kg, depending on country. Chronic and/or acute effects on sensitive species occur at copper levels occurring in some soils as a result of human activities such as copper fertiliser addition, and addition of sludge.

When soil levels exceed 150 mg Cu/kg, native and agricultural species show chronic effects. Soils in the range 500-1000 mg Cu/kg act in a strongly selective fashion allowing the survival of only copper-tolerant species and strains. At 2000 Cu mg/kg most species cannot survive. By 3500 mg Cu/kg areas are largely devoid of vegetation cover. The organic content of the soil appears to be a key factor affecting the bioavailability of copper.

On normal forest soils, non-rooted plants such as mosses and lichens show higher copper concentrations. The fruiting bodies and mycorrhizal sheaths of soil fungi associated with higher plants in forests often accumulate copper to much higher levels than plants at the same site. International Programme on Chemical Safety (IPCS): Environmental Health Criteria 200

For alkenes (olefins)

Environmental fate:

Ecotoxicity studies conducted with a wide range of products have shown little potential for toxicity to aquatic organisms under expected conditions of use or in the event of an accidental release. Not all alpha olefins are readily biodegradable; however, they will ultimately biodegrade. While the octanol/water partition coefficients of alpha olefins suggest a potential for bioaccumulation of these materials in aquatic organisms, the volatility of these materials (especially for the liquid alpha olefins) and the low-water solubility (indicative of limited bioavailability), would indicate that bioaccumulation will not occur. Under most environmental scenarios, extensive evaporation and subsequent degradation in the atmosphere would preclude bioaccumulation. Therefore, alpha olefins are not expected to be toxic to aquatic organisms, will biodegrade, and will not bioaccumulate.

The potential for exposure of aquatic organisms to members of the higher olefins will be influenced by their physico-chemical properties. The predicted or measured water solubilities of these olefins range from 50 mg/L at 20 C for hexene to 0.00015 mg/L at 25 C for 1-octadecene, and to 6.33 [E-23] mg/L at 25 C for C54 alpha olefin, which suggests there is a lower potential for the larger olefins to be bioavailable to aquatic organisms due to their low solubilities. Their vapor pressures range from 230.6 hPa at 25 C for hexene to 0.00009 hPa at 25 C for 1-octadecene, and to 1.13 [E-16] hPa at 25 C for C54 alpha olefin, which suggests the shorter chain olefins will tend to partition to the air at a significant rate and not remain in the other environmental compartments for long periods of time; while the longer chain olefins will tend to partition primarily to water, soil or sediment, depending on water solubility and sorption behavior. The predicted soil adsorption coefficients (Koc) range from 149 for C6 to 230,800 for C18 and to 1.0 [E10] for C54, indicating increasing partitioning to soil/sediment with increasing carbon number. Level I fugacity modelling predicts that the C6-13 olefins would partition primarily to air, while the C16 and longer chain olefins would partition primarily to soil. Results of Level III fugacity modelling suggest that the C6-8 olefins will partition primarily to the water compartment; and, as the chain length increases beyond C10, soil and sediment become the primary compartments. These chemicals have a very low potential to hydrolyse and do not photodegrade directly. However, in the air, all members of the category are subject to atmospheric oxidation from hydroxyl radical attack, with calculated degradation half-lives of 1.8 to 4.8 hours. C6-30 olefins have been shown to degrade to an extent of approximately 8-92% in standard 28 day biodegradation tests. These results were not clearly correlated with carbon number or any other identifiable parameter; however, the weight of evidence shows that the members of the higher olefins have potential for degradation in the environment. Volatilisation from water is predicted to occur rapidly (hours to days), with Henry's Law Constants (bond method) ranging from 0.423 (C6) to 10.7 (C18), and to 2.89 [E5] (C54) atm⁻³/mol. Consideration of these degradation processes supports the assessment that these substances will degrade relatively rapidly in the environment and not persist. Based on calculated bioconcentration factors, the C6, C7, and C16 and longer chain length category members are not expected to bioaccumulate (BCF: C6 = 44-46, C7 = 236, C16 = 71-92 and >= C18 = 3.2-4.6). Although the C8-15 olefins have BCFs ranging from 313 to 2030, and Kow values ranging from 4.13 to 7.49, and thus are considered to have the potential for bioaccumulation, their physico-chemical properties and fate indicate that there would be limited environmental exposure because of volatility, biodegradability and limited solubility.

Ecotoxicity:

Data indicate that acute aquatic toxicity can be observed for C6 through the C10 olefins (C6: EC/LC50 range of 1-10 mg/L; C7-C10: EC/LC50 range of 0.1-1.0 mg/L), and that toxicity increases with increasing carbon number within that range, which is consistent with increasing Kow values (3.07 -5.12). Above a chain length of 10, toxicity is not observed within the limits of solubility. However, data indicate that chronic aquatic toxicity can be observed in the C10 olefins (EC10 = 20.0 ug/L, EC50= 28.1 ug/L, NOEC = 19.04 ug/L). Data also suggest that aquatic toxicity does not differ with bond location or presence of branching.

For butadiene:

Kow: 1.99

Koc : 72-228

Half-life (hr) air : 4.9

Henry's Pa m3 /mol: 2.57

Henry's atm m3 /mol: 7.24E-02

BCF : 19.1

Environmental fate:

The high volatility of this compound suggests that it will partition predominantly to the atmospheric compartment, where it is not expected to be adsorbed to particulate matter to any significant extent.

Terrestrial Fate: If spilled on land, 1,3-butadiene will predominately volatilise very rapidly due to its very low boiling point. Dissolved in water, it may leach through soil into ground water due to its high water solubility and low estimated soil adsorption coefficient. It will not appreciably hydrolyse but may be subject to biodegradation based on screening tests. 1,3-Butadiene is expected to volatilize rapidly from either moist or dry soil to the atmosphere. This follows from the estimated lack of any appreciable adsorption to soil, and consideration of 1,3-butadiene's calculated Henry's law constant for moist soil or its vapor pressure, 2,100 mm Hg at 25 C, for dry soil. Both values suggest a rapid rate of volatilisation from their respective media. The calculated soil adsorption coefficient of 288 suggests that 1,3-butadiene may display moderate mobility in soil. However, the expected rapid rate of volatilisation and the possibility of rapid degradation in soil suggest that there is little potential for 1,3-butadiene to leach into groundwater. Methane-utilizing bacteria isolated from the soil of an oil refinery epoxidised 1,3-butadiene under aerobic conditions

Aquatic Fate: When released into water, 1,3-butadiene will volatilise rapidly with a half-life estimated to be several hours. It will not hydrolyse appreciably, but may be subject to biodegradation, based on screening tests.

Atmospheric Fate: Butadiene is a reactive, electron-rich chemical that is expected to undergo rapid reactions with the electrophilic oxidants typically present in the atmosphere: ozone, photochemically produced hydroxyl radicals, nitrate radicals, and molecular oxygen. Among these, the most rapid reaction in the atmosphere is with photochemically produced hydroxyl radicals. The atmospheric destruction of 1,3-butadiene by photo-initiated processes has been established empirically by early studies. There are four gas-phase pathways that can destroy 1,3-butadiene in the atmosphere. Depending on local conditions, any one or all of these reactions may occur. Destruction of atmospheric 1,3-butadiene by the gas-phase reaction with photochemically produced hydroxyl radicals is expected to be the dominant photo-initiated pathway. Destruction by nitrate radicals is expected to be a significant night-time process in urban areas.

Reaction with hydroxyl radicals is the dominant removal mechanism, with an estimated half-life of several hours. Reaction with ozone and nitrate radicals may also contribute to the degradation of the chemical. Polluted urban atmospheres increase the rate of degradation somewhat during daylight hours as suggested by the detection of the highest atmospheric levels of the chemical in the early morning hours. Acetaldehyde and acrolein have been identified as products of photooxidation. Washout may contribute to removal of 1,3-butadiene from the atmosphere; however, evaporation from the rain may be rapid and the compound returned to the atmosphere relatively quickly unless it leaches into the soil.

Biodegradation: No data concerning the biodegradation of 1,3-butadiene in natural systems could be found in the literature. 1,3-Butadiene was listed in a group of chemicals which should be biodegraded by biological sewage treatment, as long as suitable acclimatization is achieved. Screening tests suggest that 1,3-butadiene may be biodegradable in the environment with 1,2-epoxybutene being a potential product .

Soil Adsorption/Mobility: The range of estimated adsorption coefficients for 1,3-butadiene from the soils and sediments is 72-228 based on its octanol/water partition coefficient or its water solubility and would therefore not be expected to appreciably adsorb in soils and sediments.

Volatilization from Water/Soil: Using the Henry's Law constant, the estimated half-life for evaporation of 1,3-butadiene from a river 1 m deep with a 1 m/sec current and a 3 m/sec wind is 3.8 hours . Due to its low boiling point, 1,3-butadiene would be expected to rapidly evaporate from soils.

Ecotoxicity:

Fish LC50 (24 h): 71.5 mg/L

1,3-Butadiene is moderately toxic to aquatic life in the short term and slightly toxic in the long term. There is not enough information to predict additional short or long-term effects of 1,3-butadiene on plants, birds, or other animals. 1,3-Butadiene is not expected to accumulate in fish. Animal studies have reported development effects such as skeletal abnormalities and decreased foetal weights, and reproductive effects, including an increased incidence of shrinkage of the ovaries and testicles. Animal studies have also reported tumours at a variety of sites from inhalation of 1,3-butadiene.

For butane:

log Kow: 2.89

Koc: 450-900

BCF: 1.9

Environmental Fate

Terrestrial Fate: An estimated Koc value of 900, determined from a log Kow of 2.89 indicates that n-butane is expected to have low mobility in soil. Volatilisation of n-butane from moist soil surfaces is expected to be an important fate process given an estimated Henry's Law constant of 0.95 atm-cu m/mole, derived from its vapor pressure, 1820 mm Hg and water solubility, 61.2 mg/l. The potential for volatilisation of n-butane from dry soil surfaces may exist based upon its vapor pressure. While volatilisation from soil surfaces is expected to be the predominant fate process of n-butane released to soil, this compound is also susceptible to biodegradation. In one soil, a biodegradation rate of 1.8 mgC/day/kg dry soil was reported.

Aquatic fate: The estimated Koc value indicates that n-butane may adsorb to suspended solids and sediment. Volatilisation from water surfaces is expected based upon an estimated Henry's Law constant Using this Henry's Law constant volatilisation half-lives for a model river and model lake are estimated to be 2.2 hours and 3 days, respectively. An estimated BCF of 33 derived from the log Kow suggests the potential for bioconcentration in aquatic organisms is moderate. While volatilisation from water surfaces is expected to be the major fate process for n-butane released to water, biodegradation of this compound is also expected to occur. In a screening study, complete biodegradation was reported in 34 days. In a second study using a defined microbial culture, it was reported that n-butane was degraded to 2-butanone and 2-butanol. Photolysis or hydrolysis of n-butane in aquatic systems is not expected to be important.

Atmospheric fate: According to a model of gas/particle partitioning of semivolatile organic compounds in the atmosphere and the vapour pressure, n-butane, is expected to exist solely as a gas in the ambient atmosphere. Gas-phase n-butane is degraded in the atmosphere by reaction with photochemically-produced hydroxyl radicals; the half-life for this reaction in air is estimated to be 6.3 days, calculated from its rate constant of 2.54x10⁻¹² cu cm/molecule-sec at 25 deg. Based on data for iso-octane and n-hexane, n-butane is not expected to absorb UV light in the environmentally significant range, >290 nm and probably will not undergo direct photolysis in the atmosphere. Experimental data showed that 7.7% of the n-butane fraction in a dark chamber reacted with nitrogen oxide to form the corresponding alkyl nitrate, suggesting nighttime reactions with radical species and nitrogen oxides may contribute to the atmospheric transformation of n-butane.

DO NOT discharge into sewer or waterways.

Persistence and degradability

Ingredient	Persistence: Water/Soil	Persistence: Air
	No Data available for all ingredients	No Data available for all ingredients

Bioaccumulative potential

Ingredient	Bioaccumulation
	No Data available for all ingredients

Mobility in soil

Ingredient	Mobility
	No Data available for all ingredients

CRC(NZ) Copper Anti-Seize Aerosol

SECTION 13 DISPOSAL CONSIDERATIONS

Waste treatment methods

Product / Packaging disposal	<p>Legislation addressing waste disposal requirements may differ by country, state and/ or territory. Each user must refer to laws operating in their area. In some areas, certain wastes must be tracked.</p> <p>A Hierarchy of Controls seems to be common - the user should investigate:</p> <ul style="list-style-type: none"> ▶ Reduction ▶ Reuse ▶ Recycling ▶ Disposal (if all else fails) <p>This material may be recycled if unused, or if it has not been contaminated so as to make it unsuitable for its intended use. If it has been contaminated, it may be possible to reclaim the product by filtration, distillation or some other means. Shelf life considerations should also be applied in making decisions of this type. Note that properties of a material may change in use, and recycling or reuse may not always be appropriate.</p> <ul style="list-style-type: none"> ▶ DO NOT allow wash water from cleaning or process equipment to enter drains. ▶ It may be necessary to collect all wash water for treatment before disposal. ▶ In all cases disposal to sewer may be subject to local laws and regulations and these should be considered first. ▶ Where in doubt contact the responsible authority. ▶ Consult State Land Waste Management Authority for disposal. ▶ Discharge contents of damaged aerosol cans at an approved site. ▶ Allow small quantities to evaporate. ▶ DO NOT incinerate or puncture aerosol cans. ▶ Bury residues and emptied aerosol cans at an approved site.
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SECTION 14 TRANSPORT INFORMATION

Labels Required

	
Marine Pollutant	NO
HAZCHEM	Not Applicable

Land transport (ADG)

UN number	1950				
UN proper shipping name	AEROSOLS				
Transport hazard class(es)	<table border="0"> <tr> <td>Class</td> <td>2.1</td> </tr> <tr> <td>Subrisk</td> <td>Not Applicable</td> </tr> </table>	Class	2.1	Subrisk	Not Applicable
Class	2.1				
Subrisk	Not Applicable				
Packing group	Not Applicable				
Environmental hazard	Not Applicable				
Special precautions for user	<table border="0"> <tr> <td>Special provisions</td> <td>63 190 277 327 344 381</td> </tr> <tr> <td>Limited quantity</td> <td>1000ml</td> </tr> </table>	Special provisions	63 190 277 327 344 381	Limited quantity	1000ml
Special provisions	63 190 277 327 344 381				
Limited quantity	1000ml				

Air transport (ICAO-IATA / DGR)

UN number	1950														
UN proper shipping name	Aerosols, flammable														
Transport hazard class(es)	<table border="0"> <tr> <td>ICAO/IATA Class</td> <td>2.1</td> </tr> <tr> <td>ICAO / IATA Subrisk</td> <td>Not Applicable</td> </tr> <tr> <td>ERG Code</td> <td>10L</td> </tr> </table>	ICAO/IATA Class	2.1	ICAO / IATA Subrisk	Not Applicable	ERG Code	10L								
ICAO/IATA Class	2.1														
ICAO / IATA Subrisk	Not Applicable														
ERG Code	10L														
Packing group	Not Applicable														
Environmental hazard	Not Applicable														
Special precautions for user	<table border="0"> <tr> <td>Special provisions</td> <td>A145 A167 A802</td> </tr> <tr> <td>Cargo Only Packing Instructions</td> <td>203</td> </tr> <tr> <td>Cargo Only Maximum Qty / Pack</td> <td>150 kg</td> </tr> <tr> <td>Passenger and Cargo Packing Instructions</td> <td>203</td> </tr> <tr> <td>Passenger and Cargo Maximum Qty / Pack</td> <td>75 kg</td> </tr> <tr> <td>Passenger and Cargo Limited Quantity Packing Instructions</td> <td>Y203</td> </tr> <tr> <td>Passenger and Cargo Limited Maximum Qty / Pack</td> <td>30 kg G</td> </tr> </table>	Special provisions	A145 A167 A802	Cargo Only Packing Instructions	203	Cargo Only Maximum Qty / Pack	150 kg	Passenger and Cargo Packing Instructions	203	Passenger and Cargo Maximum Qty / Pack	75 kg	Passenger and Cargo Limited Quantity Packing Instructions	Y203	Passenger and Cargo Limited Maximum Qty / Pack	30 kg G
Special provisions	A145 A167 A802														
Cargo Only Packing Instructions	203														
Cargo Only Maximum Qty / Pack	150 kg														
Passenger and Cargo Packing Instructions	203														
Passenger and Cargo Maximum Qty / Pack	75 kg														
Passenger and Cargo Limited Quantity Packing Instructions	Y203														
Passenger and Cargo Limited Maximum Qty / Pack	30 kg G														

Sea transport (IMDG-Code / GGVSee)

UN number	1950
UN proper shipping name	AEROSOLS

CRC(NZ) Copper Anti-Seize Aerosol

Transport hazard class(es)	IMDG Class	2.1
	IMDG Subrisk	Not Applicable
Packing group	Not Applicable	
Environmental hazard	Not Applicable	
Special precautions for user	EMS Number	F-D , S-U
	Special provisions	63 190 277 327 344 381 959
	Limited Quantities	1000 ml

Transport in bulk according to Annex II of MARPOL and the IBC code

Not Applicable

SECTION 15 REGULATORY INFORMATION

Safety, health and environmental regulations / legislation specific for the substance or mixture

PARAFFINIC DISTILLATE, HEAVY, SOLVENT-DEWAXED (SEVERE) IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)

Chemical Footprint Project - Chemicals of High Concern List
International Agency for Research on Cancer (IARC) - Agents Classified by the IARC Monographs

GRAPHITE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)

COPPER IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Inventory of Chemical Substances (AICS)
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 5
Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 6

HYDROCARBONS, C3-4 RICH, PETROLEUM DISTILLATE IS FOUND ON THE FOLLOWING REGULATORY LISTS

Australia Hazardous Chemical Information System (HCIS) - Hazardous Chemicals
Australia Inventory of Chemical Substances (AICS)

Australia Standard for the Uniform Scheduling of Medicines and Poisons (SUSMP) - Schedule 4
Chemical Footprint Project - Chemicals of High Concern List

National Inventory Status

National Inventory	Status
Australia - AICS	Yes
Canada - DSL	Yes
Canada - NDSL	No (paraffinic distillate, heavy, solvent-dewaxed (severe); graphite; copper; hydrocarbons, C3-4 rich, petroleum distillate)
China - IECSC	Yes
Europe - EINEC / ELINCS / NLP	Yes
Japan - ENCS	No (graphite; copper; hydrocarbons, C3-4 rich, petroleum distillate)
Korea - KECI	Yes
New Zealand - NZIoC	Yes
Philippines - PICCS	No (hydrocarbons, C3-4 rich, petroleum distillate)
USA - TSCA	Yes
Taiwan - TCSI	Yes
Mexico - INSQ	No (hydrocarbons, C3-4 rich, petroleum distillate)
Vietnam - NCI	Yes
Russia - ARIPS	No (hydrocarbons, C3-4 rich, petroleum distillate)
Legend:	Yes = All CAS declared ingredients are on the inventory No = One or more of the CAS listed ingredients are not on the inventory and are not exempt from listing(see specific ingredients in brackets)

SECTION 16 OTHER INFORMATION

Revision Date	01/11/2019
Initial Date	12/03/2013

SDS Version Summary

Version	Issue Date	Sections Updated
3.1.1.1	03/10/2017	Acute Health (inhaled), Acute Health (skin), Chronic Health, Classification, Physical Properties, Spills (major), Storage (storage requirement)
5.1.1.1	01/11/2019	One-off system update. NOTE: This may or may not change the GHS classification

Other information

Classification of the preparation and its individual components has drawn on official and authoritative sources as well as independent review by the Chemwatch Classification

Continued...

committee using available literature references.

The SDS is a Hazard Communication tool and should be used to assist in the Risk Assessment. Many factors determine whether the reported Hazards are Risks in the workplace or other settings. Risks may be determined by reference to Exposures Scenarios. Scale of use, frequency of use and current or available engineering controls must be considered.

Definitions and abbreviations

PC—TWA: Permissible Concentration-Time Weighted Average
PC—STEL: Permissible Concentration-Short Term Exposure Limit
IARC: International Agency for Research on Cancer
ACGIH: American Conference of Governmental Industrial Hygienists
STEL: Short Term Exposure Limit
TEEL: Temporary Emergency Exposure Limit.
IDLH: Immediately Dangerous to Life or Health Concentrations
OSF: Odour Safety Factor
NOAEL :No Observed Adverse Effect Level
LOAEL: Lowest Observed Adverse Effect Level
TLV: Threshold Limit Value
LOD: Limit Of Detection
OTV: Odour Threshold Value
BCF: BioConcentration Factors
BEI: Biological Exposure Index

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